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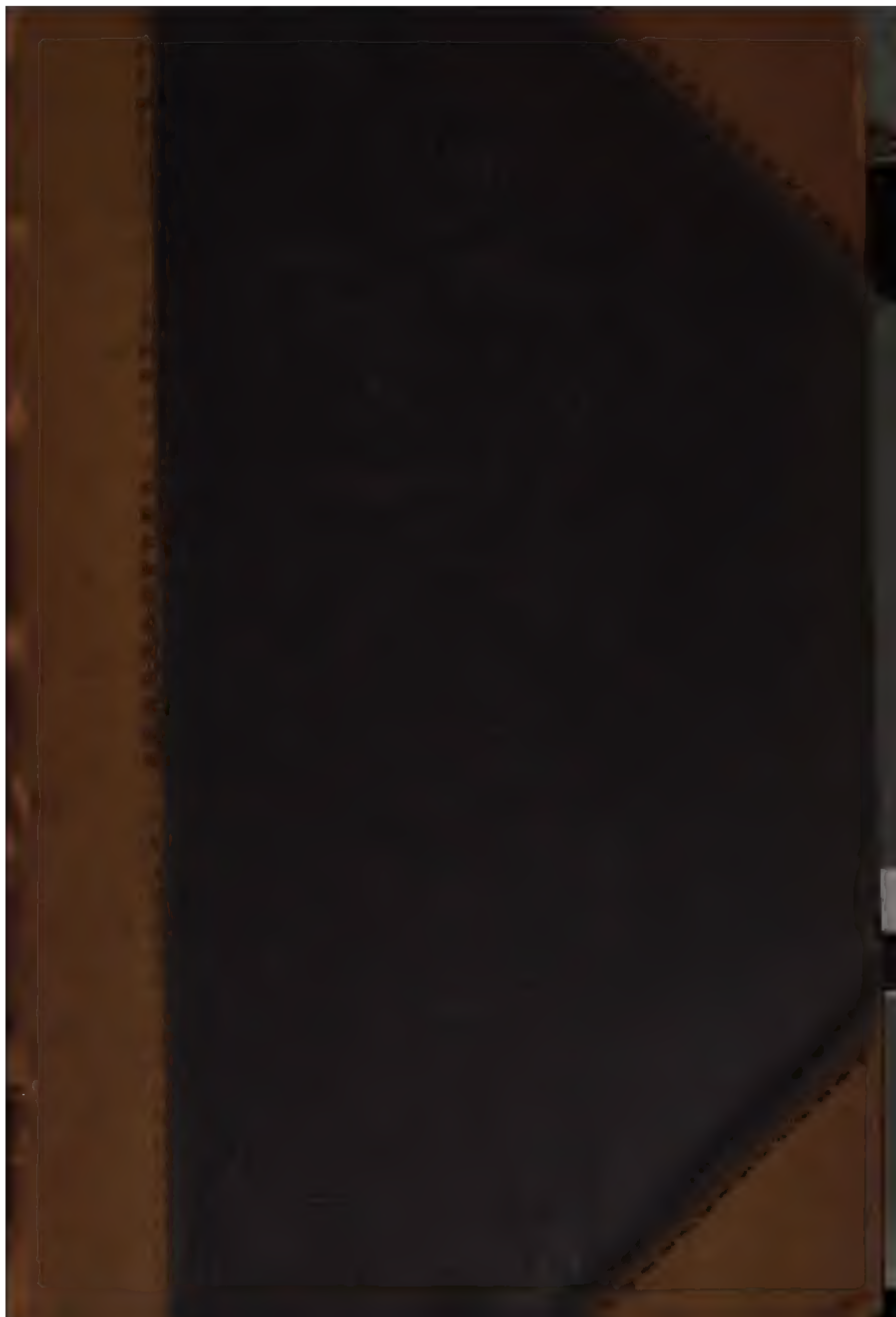
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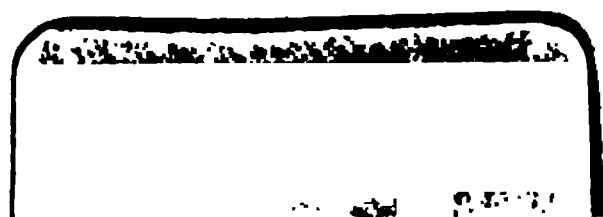
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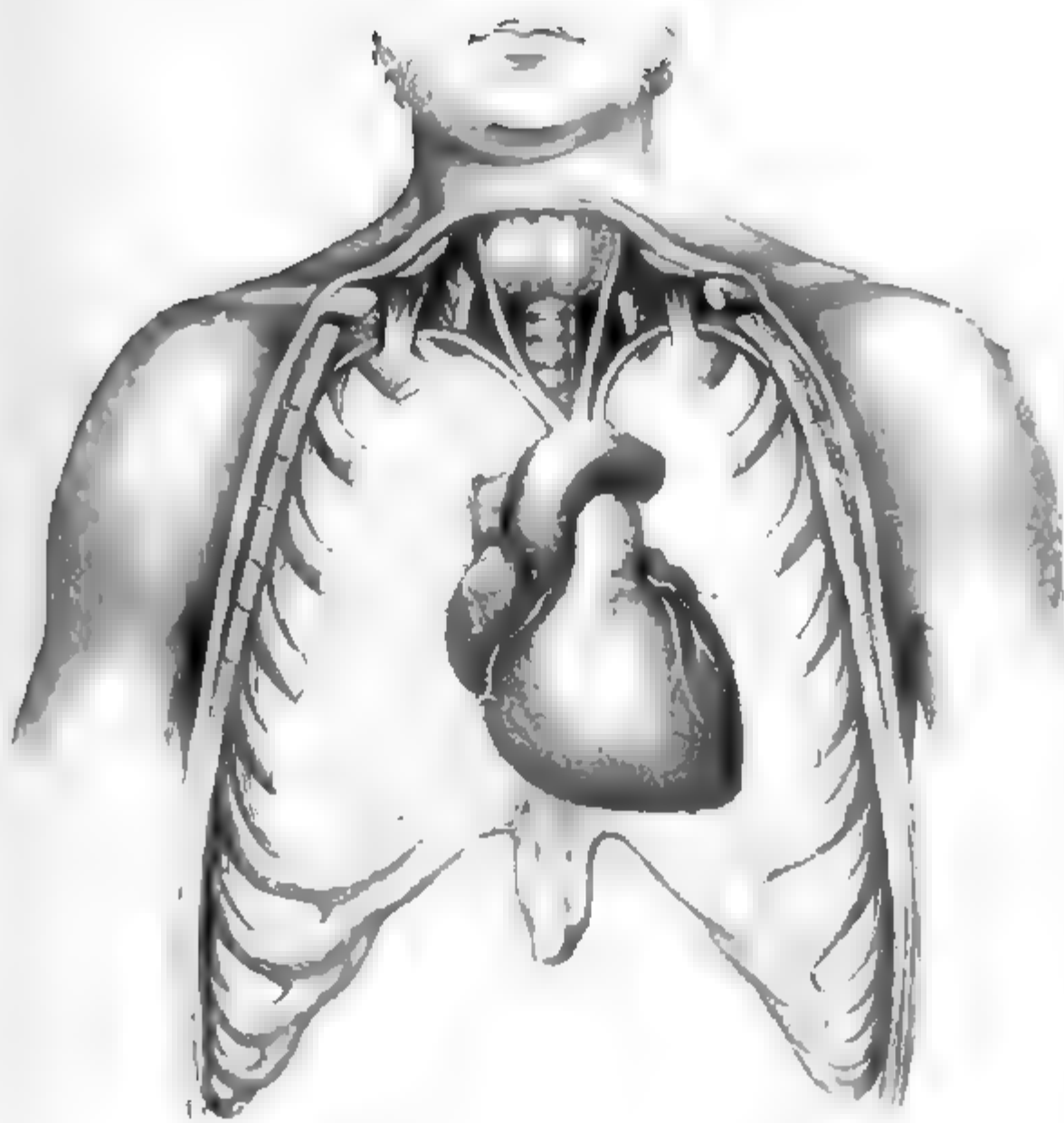
**A TREATISE**  
**ON**  
**DISEASES OF THE HEART.**











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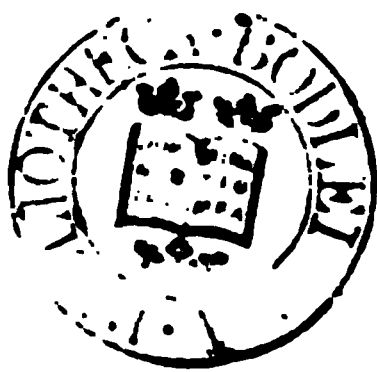
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**SOCIETY OF IRELAND, ETC.**

**"lenire dolorem**  
**Possis, et magnam morbi deponere partem."**

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TO

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**SEVERAL TREATISES ON OPHTHALMIC SURGERY,**

**PROPRIETOR OF**

**The Dublin Medical Press,**

**THE FOLLOWING PAGES ARE INSCRIBED,**

**AS A**

**TRIFLING TESTIMONY OF THE AUTHOR'S ESTIMATION OF HIS TALENTS;**

**NO LESS THAN TO MARK HIS SENSE OF**

**THE SERVICES RENDERED BY DR. JACOB TO THE MEDICAL PROFESSION,**

**THROUGHOUT IRELAND, IN FOUNDING AND CONDUCTING THE**

**FIRST IRISH WEEKLY MEDICAL PERIODICAL,**

**AND HIS OPINION OF THE**

**ABILITY AND ENERGY WITH WHICH THE RIGHTS AND PRIVILEGES OF THE**

**MEDICAL PROFESSION HAVE BEEN INVARIABLY ADVOCATED**

**BY DR. JACOB IN THE PAGES OF**

**THAT JOURNAL.**



## PREFACE.

---

THE diseases which constitute the subject matter of this treatise are sufficiently frequent, and sufficiently distressing, to have always attracted a large share of the attention of the Profession ; it is only, however, since the discovery of auscultation that they admitted of being diagnosed with accuracy ; or, that the symptoms which characterize each, admitted of being laid down with precision ; and the School of Dublin may claim some share of the credit of having brought about this desirable result.

The present treatise is, in a great measure, founded upon Clinical Lectures delivered at different periods in St. Vincent's Hospital ; some of these lectures appeared at the time in the pages of the "Dublin Medical Press," others in the "London Medical Gazette ;" they were favourably received by the profession ; and the author having been invited to publish them in a distinct form, has carefully revised them, added much additional matter, and re-written a considerable portion.

The work consists of two Parts : the first contains a description of the healthy heart, its size, weight, and the

measurements of its chambers and orifices ; followed by sufficiently full details respecting its motions and sounds. The examination of the heart in disease is then entered upon, commencing with the physical signs ; the general signs, and the secondary or remote symptoms of cardiac disease are described in succession.

The second part is devoted to the individual diseases of the heart, which are arranged according as their seat is the investing membrane, the lining membrane, or the muscular tissue of the heart, followed by a description of the functional or inorganic affections of the heart.

The author's object in this publication has been to give a concise, though sufficiently complete, description of the heart in health, and disease. No point of any practical importance has been omitted that he is aware of ; and he has endeavoured, as far as lay in his power, to assign the credit of every original discovery or improvement, in theory or practice, to its legitimate source.

*Kildare-street, May, 1853.*



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ON

# DISEASES OF THE HEART.

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## CHAPTER I.

### EXAMINATION OF THE HEART IN HEALTH—DESCRIPTION OF THE SEVERAL PARTS OF THE HEART.

THE Heart, the central organ of the circulation by which the blood is transmitted to the lungs, and to the remotest parts of the body, and to which the blood from the lungs and from every part of the system is returned, is a hollow muscular viscus, divided in its interior into several compartments, covered externally and lined throughout by distinct membranes; situated in the middle mediastinum, near the centre of the cavity of the thorax, about the junction of the superior with the two inferior thirds of the body; lying between the lungs and the two layers of pleura which constitute the mediastinum, and enclosed in a proper fibrous capsule, the pericardium.

The heart occupies the first place in the circulatory system; its functions yield in importance to those of no other organ, for life is dependent upon its regular and constant action, and the healthy exercise of the functions of every other organ mainly depends upon its integrity. Hence its physiology and pathology have always constituted subjects of the highest interest to the medical inquirer, and its diseases have occupied the pen of some of the brightest ornaments of the medical profession.

Although much valuable information, upon various points connected with our subject, existed previous to the discovery of auscultation, much, also, it must be confessed, remained unknown. Laennec's beautiful discovery opened up an altogether new means of investigating the diseases of this organ ; it added, in fact, a new sense to our other methods of detecting and distinguishing between them ; and it gave an impulse to the study of diseases of the thoracic viscera which has continued unabated to the present day. So that there are scarcely any other organs the physiology and pathology of which have made more rapid advances, and the present state of our knowledge of which so remarkably contrasts with that which prevailed at a period comparatively recent.

If it be true, as a general rule, that abnormal conditions of organs cannot be recognised or distinguished without a knowledge of what constitutes their healthy state, and that derangement of function cannot be explained without an acquaintance with the functions in health, to no organ does this so peculiarly or with such force apply as to the heart ; and yet, until within a few years, we had little positive information upon points apparently so simple as the size and weight of the healthy heart : erroneous opinions likewise prevailed respecting the thickness of the walls, the capacity of the cavities, and the diameter of the orifices of the healthy heart ; and nothing certain was known as to the mechanism by which its sounds are produced.

It is obvious that unless correct ideas on these heads are entertained, we shall not be in a position to decide whether the parietes of the heart are hypertrophied or attenuated ; whether its chambers are dilated or preserve their normal dimensions ; or whether its orifices are increased or diminished in diameter. Unless the exact size and shape and position of the heart are known, deviations from its normal condition are liable to be overlooked, or functional derangement to be mistaken for organic disease ; and unless we are familiar with the normal sounds of the heart, with the situation at which they are best heard, and with the mechanism by which each is produced, we shall not be in a position to recognise abnormal sounds, much less to trace them to their source, or to determine their cause.

I propose, therefore, previous to entering upon diseases of the heart, to consider briefly the anatomy and physiology of this

organ ; to describe its exact position, and the relation of its several parts to points upon the surface, and to give a summary of the researches which have been made respecting its weight and size, the thickness of its parietes, the capacity of its cavities, and the diameter of its orifices.

*Shape of the Heart.*—The shape of the heart is almost too well known to require description ; the older anatomists compared it to a pyramid, or to a flattened cone ; but a flattened cone (as Winslow remarked) is not a cone. The heart, taken as a whole, has not a regular geometrical figure ; and as the term *cordiform* has been generally adopted, and is familiar to most persons, it will be sufficient to say that the heart has a cordiform shape, the auricles forming the upper and broader part, the ventricles the lower, narrower, and more conical part ; this conical form of the ventricular portion of the organ being produced by the peculiar arrangement of the muscular fibres which go to form its walls.

The base of the heart is formed by the auricles, its apex by the left ventricle alone, as the right ventricle does not descend as low as the left. The anterior surface of the ventricular portion of the heart is convex ; the posterior surface, which rests upon the diaphragm, is flattened. The left border or edge of the heart is obtuse and rounded ; the right edge is more acute.

*Divisions of the Heart.*—The heart, although apparently a single organ, is in reality double, and consists of two symmetrical hearts joined together, each with its own system of vessels, each circulating a different kind of blood, and each performing a distinct office ; the one circulating the blood through the lungs, the other through the system ; the one receiving and transmitting venous, the other arterial blood, but both simultaneously performing their functions.

One of these divisions of the heart is termed the *right*, the other the *left* side of the organ ; but from their relative position, the former should rather (as was long since pointed out by Winslow) be termed the anterior, the latter the posterior side of the heart. These terms are retained from the earlier anatomists, having been applied at a period when the knowledge of anatomy was derived chiefly from the dissection of animals, in which the heart lies vertically in the chest, not obliquely as in the human subject. Each of these divisions of the heart consists of two

cavities, an auricle and a ventricle, which communicate with one another but have no direct communication with their corresponding cavities : the auricle on both sides is above, the ventricle below.

The parietes of the right side of the heart, particularly of the ventricular portion, are much thinner than those of the left ; the right ventricle has merely to transmit its blood through the lungs, while the left has to transmit its blood throughout the system. The pulmonary artery, likewise, has thinner coats and more delicate valves than the aorta, evidently because so much power of resistance was not required.

The right side of the heart, owing to its receiving and transmitting dark or venous blood, was named by Bichat, “cœur à sang noir ;” the left side, from its receiving and transmitting red or arterial blood, was termed by him, “cœur à sang rouge.” The right auricle is sometimes termed the sinus of the venæ cavæ ; the left, the sinus of the pulmonary veins. The right ventricle is sometimes termed the pulmonary ventricle ; the left, the aortic or systemic ventricle.

*Circular and longitudinal Grooves.*—The distinction between these parts is marked by grooves upon the surface of the heart ; two of which are longitudinal, and one circular. The anterior longitudinal groove runs from the base of the ventricles to the right side of the apex of the heart, dividing this part into two unequal halves : in it, the principal division of the anterior or left coronary artery is lodged. Upon the posterior surface, a similar groove, which divides the ventricular portion of the heart into two nearly equal parts, lodges a large branch of the posterior or right coronary artery. These grooves, which are often in a great measure obliterated by adipose tissue, mark pretty exactly the site of the septum ventriculorum. In some instances, owing to the anterior longitudinal groove being continued through the apex, this part has the appearance of being double or forked. Morgagni found this well marked in five out of eighteen subjects, which he examined for the purpose. The circular groove, which runs round the heart between the base of the ventricles and the auricles, lodges the trunk of the posterior or right coronary artery, and a large branch of the anterior or left coronary artery : it marks the line of separation between the auricles and the ventricles.

The fat which accumulates upon the surface of the heart is



always first deposited in these grooves: in the circular groove first; next in the anterior; and next in the posterior longitudinal groove: when a larger amount occurs, it will be found along the thin edge of the right ventricle, and upon the margin of the appendices of the auricles, particularly the left. It is usually met with in persons who have a general tendency to obesity; but a considerable amount is occasionally found in subjects dying of chronic disease, accompanied by the absorption of all the subcutaneous fat. Fat is altogether absent in the heart of the infant, and M. Bizot has shown that sex has some influence upon its deposition, as it is more frequent in the female than the male; and this holds good even in diseases accompanied by much emaciation; thus in eleven males who died of phthisis, fat was completely absent in six; while in twenty-six females who died of the same disease, it was completely absent in only three.

*Position of the Heart.*—In order to determine the exact and the relative position of the heart, the most eligible plan to adopt is to dissect away the soft parts from the front of the chest, with the intercostal muscles, in a subject where the parts are perfectly healthy: the pericardium being then laid open, the position of the heart, and the relation of its several parts to fixed points upon the surface, can be readily determined.

The heart is situated obliquely in the cavity of the thorax from above downwards, from before backwards, and from right to left: it lies behind the middle and lower bone of the sternum, also behind the cartilages of the 3rd, 4th, and 5th right ribs near the sternum, and the cartilages of the 3rd, 4th, 5th, and 6th ribs on the left side, in front of the bodies of the 6th, 7th, and 8th dorsal vertebræ, and immediately above the diaphragm, upon the cordiform tendon of which it rests, the serous layer of the pericardium only being interposed.

Owing to its oblique position, the base of the heart looks upwards and backwards towards the right shoulder, its apex downwards and forwards towards the space between the cartilages of the 5th and 6th ribs on the left side, where its impulse may be felt during life. In the early period of intra-uterine life the heart lies vertically in the chest, as in mammalia generally: it is not until the beginning of the fourth month that it commences to assume the position which it afterwards retains.

*Relations of the Heart.*—In the healthy chest, the heart is overlapped laterally, and in a great measure in front, by the lungs; posteriorly the œsophagus and descending aorta lie between it and the bodies of the dorsal vertebræ. Inferiorly, the heart rests upon the cordiform tendon of the diaphragm, the serous layer of the pericardium only being interposed. The base of the heart is on a line with the interval between the cartilages of the 2nd and 3rd ribs: its apex is a little below the 5th left rib, slightly to the left of the junction of this rib with its cartilage. During life, its impulse is felt between the cartilages of the 5th and 6th left ribs, at a point about two inches below the nipple (in the male), and three inches to the left of a vertical line through the centre of the sternum; the nipple in the adult male being immediately opposite the lower edge of the fourth left rib, a little more than an inch to the left of the junction of this rib with its cartilage.

*Region of the Heart's superficial dulness.*—The anterior border of the lungs in the healthy subject corresponds above to a vertical line through the centre of the sternum: about the middle of this bone, and pretty nearly on a line with the cartilage of the fourth ribs, the margins of the opposite lungs begin to separate from one another, the line of the left being much more oblique than that of the right. Thus, a small portion of the heart's surface is uncovered by lung, which has a triangular shape, the base below, the apex above: it consists of a portion of the apex of the right ventricle, and part of the left ventricle near its apex. This triangular space is seated on a plane below the nipple and the fourth rib: its base is on a line with the cartilage of the sixth ribs; its right boundary is nearly a vertical line through the centre of the sternum; its left an oblique line through the cartilages of the fifth and sixth ribs on the left side. This is the part of the præcordial region where the heart is in contact with the parietes of the chest, and where a dull sound is yielded by percussion.

The relations of the lungs to the heart, and consequently the amount of the surface of the heart which comes in contact with the parietes of the chest, are altered, it must be recollected, by respiration, particularly by a forced inspiration or expiration: they are likewise materially altered by disease. On a full inspiration the lungs expand, and the heart is more overlapped by these organs;

at the same time the contraction of the muscular fibres of the diaphragm draws downwards its central tendon (to which the pericardium is attached), and with it the heart: hence the region of the heart's dulness will be both diminished laterally and will be on a plane lower down than natural. "The deepest possible inspiration, however, even in cases of emphysema, never (according to Dr. Sibson) obliterates the region of the heart's dulness." "It is often, under such circumstances, quite below the sternum, and behind the xyphoid cartilage, and the cartilages of the 6th, 7th, and 8th left ribs." On a full expiration, the volume of the lungs is diminished, less of the heart's surface is overlapped by them, and the region of the heart's dulness will be enlarged laterally; while, owing to the ascent of the diaphragm, and the diminished volume of the lungs, it will be on a plane somewhat higher than natural. In persons with narrow chests, the region of the heart's dulness is almost always greater than in individuals with broad and expanded chests.

*Connexions of the Heart.*—The heart, I have said, rests upon the diaphragm: it is connected to it by the fibrous layer of the pericardium which is inserted into the cordiform tendon, as well as by the serous layer which lines the central aponeurosis of the diaphragm where the fibrous layer is deficient. It is still further connected to it by the ascending vena cava; the orifice in the tendinous portion of the diaphragm, by which this vein passes from the abdomen to the thorax, is close to the junction of the inferior cava with the right auricle of the heart; and the tendinous fibres which come off from the margin of the foramen in the diaphragm proceed upwards and downwards upon the anterior, posterior, and lateral surfaces of the vein, and unite the two parts very closely together. The opening in the diaphragm, through which the ascending cava passes, corresponds to the upper part of the xyphoid cartilage: it is on a line with the cartilage and lower margin of the fifth ribs in front, and with the body of the ninth dorsal vertebra posteriorly.

The heart is retained *in situ* by the pericardium, by the two layers of pleura which constitute the mediastinum, and by the great vessels which convey the blood to and from it; above, the aorta and pulmonary artery proceed from the base of the ventricles; posteriorly, upon each side of the left auricle, the pulmonary veins

open into it; on the right side, posteriorly, the ascending and descending venæ cavæ open into the right auricle; the former vein, as I have said, being intimately united to the diaphragm.

The base of the heart, from its connexion with the great vessels, is in a great measure fixed: the apex and body of the ventricular portion are free, but the motions of this part are limited by the pericardium. The whole organ is, however, liable to slight changes of position during inspiration or expiration, and in the erect or recumbent posture: during inspiration the heart descends slightly; during expiration it ascends slightly; in the recumbent posture the heart recedes from the parietes of the thorax; in the erect posture it approaches them. In diseased states of the lungs or pleura, the heart may be displaced considerably: thus, when a large amount of fluid is effused into the pleura on one side, the heart will be pushed towards the opposite side: in emphysema of the lungs of long standing, the heart is protruded downwards; while in large abdominal tumors, in ascites, pregnancy, &c., it will be pushed upwards. In cases of large introthoracic tumors, the heart may be displaced either upwards, downwards, forwards, backwards, or laterally, according to the situation and size of the tumor, and the rapidity of its growth. These matters will, however, be fully considered when we come to describe the diseased states of the organ.

#### CHAMBERS OF THE HEART.

The chambers of the heart are four in number—two auricles, and two ventricles. The auricles are two musculo-membranous sacs, which occupy the upper and posterior part of the organ, forming its base, separated from one another by a thin musculo-membranous septum; the ventricles are two muscular cavities, which form the body and apex of the heart, separated from one another by a thick muscular septum. The auricle and ventricle upon each side communicate with one another by a large orifice, through which the blood can pass readily from the former into the latter cavity, but is prevented from returning by a valvular apparatus. The right auricle receives the venous blood from the system generally, from which it passes into the right ventricle, by which it is propelled through the lungs. The left auricle receives the arterialized blood from the lungs, from which it passes into

the left ventricle, by which it is propelled through the system. The auricles and ventricles contract and dilate alternately, and a double circulation is thus constantly going on—a lesser and a greater, or a pulmonic and systemic, and the blood traverses two circles in its course—a greater and a lesser, which are continuous the one with the other.

The organs which belong to the greater or systemic circulation are the left ventricle, the aorta and its branches, the veins of the body, and the right auricle. The organs which belong to the lesser or pulmonic circulation are the right ventricle, the pulmonary artery and veins, and the left auricle. The greater or systemic circulation commences in the minute ramifications of the pulmonary veins, and terminates in the capillaries of the arteries of the body. The lesser or pulmonic circulation begins in the capillaries of the veins of the body, and ends in the ultimate ramifications of the pulmonary artery. Thus the blood which has served the purposes of nutrition is brought by the *venæ cavæ* to the right side of the heart, by which it is transmitted to the lungs, where it is submitted to the action of atmospheric air and assumes the bright red colour of arterial blood. It is then returned by the pulmonary veins to the left side of the organ, to be again distributed throughout the system.

*Mode of opening the Heart.*—In order to display the interior of the ventricles, and to permit of the examination of the valves, some care is required in opening the heart. The most simple and most convenient method appears, to me, to be to commence the incision in the pulmonary artery, carrying it through this vessel into the right ventricle, cutting between the sigmoid valves, and continuing it to the apex, keeping close to the septum. A transverse incision, meeting the other about its centre, may then be made in the walls of the right ventricle. The aorta being next separated from the pulmonary artery, a similar incision is to be made through it into the left ventricle, and continued to its apex, keeping close to the septum. This incision can then be continued on through the apex upon the posterior surface of the heart. To display the interior of the right auricle a crucial incision may be made upon its anterior aspect, while the left auricle may be opened upon its posterior aspect, between the orifices of the opposite pulmonary veins.

*Right Auricle.*—The right auricle forms the right, the inferior, and a portion of the anterior surface of the base of the heart. Its anterior surface lies to the right of the sternum; its posterior surface rests upon the diaphragm. It consists, in general terms, of a sinus, and an auricular appendix. The sinus of the auricle receives the blood from the descending and ascending venæ cavæ; the auricular appendix, which lies behind the cartilage of the third right rib, its tip resting against the right side of the ascending portion of the arch of the aorta, pretty nearly on a line with the sigmoid valves of the pulmonary artery, has a rude resemblance to the ear of a dog, from which it received the name, which has come since to be applied to the entire chamber. It varies somewhat in size and shape in different subjects. Its margin is often notched or serrated, something like the edge of the comb of a cock (to which the earlier anatomists compared it). This appearance is, however, usually better marked in the appendix of the left than the right auricle.

The capacity of the cavity of the right auricle exceeds that of the left (as first pointed out by Vesalius): its parietes are also thinner. Between the orifice of the superior cava and the appendix of the auricle a thick fasciculus of muscular fibres projects upon the interior, from which smaller fasciculi come off at a right angle, and run parallel to one another towards the appendix. The latter have received the name of *musculi pectinati*, from their fancied resemblance to the teeth of a comb, the thick fasciculus before mentioned forming its back. This is more evident in hearts in which the walls of the auricle are hypertrophied. In the auricular appendix the adjoining fasciculi are connected together by transverse branches, which gives its interior a reticulated appearance. In the interstices of these fasciculi the parietes of the auricle, when put on the stretch, will be observed to be semitransparent in some places, the pericardium and endocardium being at these points in contact, and alone forming its walls.

On its right side posteriorly the right auricle receives the blood from the descending or superior, and the ascending or inferior vena cava. The former returns the blood from the head and upper extremities, and opens into the superior and anterior part of the auricle; the latter, which is on a plane posterior to it, returns the blood from the abdomen and lower extremities, and

opens into the lower and back part of the auricle, near the septum auricularum. The manner in which the two venæ cavæ communicate with the auricle is such, that the blood from the superior cannot fall into the orifice of the inferior cava. The current of the superior cava is downwards and forwards towards the auriculo-ventricular orifice: the current of the inferior cava is backwards and to the left side, towards the septum of the auricles, and it enters the auricle nearly at a right angle with the rest of the vein, immediately above the Eustachian valve, which directs the current towards the fossa ovalis.

The left posterior wall of this auricle is formed by the septum auricularum, at the lower part of which, immediately above the orifice of the inferior cava, we see the remains of the foramen ovale, through which, during foetal life, the blood passed from the right to the left auricle, particularly that conveyed by the ascending vena cava, which is directed towards this orifice by the Eustachian valve. It is by no means rare to find the foramen ovale only partially obliterated in hearts otherwise perfectly healthy, and in subjects who had never exhibited any symptom of morbus cœruleus. The opening is usually valvular, is seated at its upper part, and will frequently admit a probe or the handle of a scalpel. M. Bizot states, that he found the foramen ovale in the same condition in which it is at birth, or only partially obliterated, in eighteen out of seventy-three male subjects, and in twenty-six out of eighty-two female.

*Right auriculo-ventricular Orifice.*—Inferiorly the auricle is united to its corresponding ventricle, and here the orifice of communication between the auricle and the ventricle is seated. This, the right auriculo-ventricular orifice, has an elliptic shape, the long diameter from before backwards, as the heart lies *in situ*: it is larger than the corresponding orifice of the left side, and its margin, like that of the latter, consists of dense, white, fibrous or tendinous tissue. By the older anatomists this was termed the right tendon of the heart. “It is plainly,” Bell observes, “the place of union between the auricle and the ventricle, which are in the foetus (the chick, for example) distinct bags.” The right auriculo-ventricular orifice is situated behind the sternum, pretty nearly on a line with the junction of the cartilages of the fourth ribs with that bone. Between the Eustachian valve and the



auriculo-ventricular orifice the opening of the coronary vein is situated, provided with a valve formed by a semilunar fold of the lining membrane, which partially closes its orifice, and prevents regurgitation into it.

*Right Ventricle.*—The right or pulmonic ventricle forms the right anterior and inferior portion of the body of the heart. The greater part of it lies behind the sternum. On the left side of this bone it extends under the sternal extremity of the cartilages of the third, fourth, fifth, and sixth left ribs: on the right side of this bone a small portion of it extends under the cartilages of the fourth and fifth right ribs, close to the sternum. Its inferior margin is about on a line with the junction of the xyphoid cartilage and the sternum. The base of the right ventricle is connected to the auricle of the same side: its apex is a little above the apex of the heart: its anterior wall, which lies immediately under the sternum, is convex: its inferior wall, which rests upon the diaphragm, is flattened: its left or posterior wall is formed by the septum ventriculorum.

The parietes of the right ventricle are much thinner, its columnæ carneæ are smaller, and its cavity (as was first pointed out by Vesalius) is somewhat larger than that of the left ventricle: this has been supposed to depend upon its parietes being more yielding than those of the left, and from the blood accumulating in, and distending it after death; but accurate measurement has shown that its cavity exceeds the left in capacity. The right ventricle has not a regular or symmetrical shape—it is broader, but not so long as the left; the septum, which is convex towards its cavity, belongs more to the left than the right ventricle: and as Bell remarked, “the right ventricle seems to be wrapped round the left,” while the latter alone forms the apex of the heart. The right ventricle ascends higher than the left: hence the orifice of the pulmonary artery is on a plane higher than that of the aorta.

The cavity of the right ventricle consists of two parts, as first particularly described by Lieutaud—viz. an *arterial* and an *auricular* portion, separated from one another by a prominent bundle of muscular fibres, and by the largest division of the tricuspid valve. The arterial portion—the “*conus arteriosis*” of Wolff, the “*infundibulum*” of Cruveilhier—is very smooth upon its surface,



leads towards the pulmonary artery and is prolonged upwards above the level of the rest of the ventricle; in it the current is established by the systole of the ventricle, which is continuous with that of the pulmonary artery. The auricular portion, on the other hand, is very irregular upon its surface, owing to numerous prominent bundles of muscular fibres (*carneæ columnæ*) which exist here; it receives the blood directly from the right auricle, and into it there is a current through the auriculo-ventricular orifice until the closure of the tricuspid valve. Mr. Serle, in his account of the arrangement of the muscular fibres of the heart, describes the cavity of the right ventricle as consisting of three parts: an "auricular," a "pulmonary or ventricular," and an "apical;" the pulmonary and apical form the arterial portion of Lieutaud. "The pulmonary (according to him) is that formed by the fibres which arise from the root of the pulmonary artery at its entire circumference: the apical channel is that which forms the channel of communication between the other two, and which extends to the apex."

The *carneæ columnæ* are very numerous in the auricular portion of the ventricle: many run from the apex towards the base, others cross them, leaving deep sulci between them: some of these fleshy columns are attached in their whole length, others only by their extremities, and others again only by one extremity, the opposite giving insertion to the *chordæ tendineæ* of the tricuspid valve: the latter, which are sometimes termed *musculi papillares*, are fewer in number than either of the other kinds.

At the base of the ventricle two orifices are situated: one (the auriculo-ventricular orifice) communicates with the auricle on that side; the other is the orifice of the pulmonary artery: each is provided with a valvular apparatus. The auriculo-ventricular orifice is situated at the posterior and right side of the base of the ventricle; the pulmonary orifice is to the left side, immediately above the septum of the ventricles, on a plane anterior to, and three-quarters of an inch higher up, than the other.

*Tricuspid Valve.*—The right auriculo-ventricular orifice is encircled by a valve, formed by a duplicature of the lining membrane, strengthened by fibrous tissue, which comes off from the margin of the orifice: the free extremity of this valve hangs down into the ventricle, where, by means of the *chordæ tendineæ*

it is connected to the *carneæ columnæ*, and so with the walls of the ventricle. This valve is termed *tricuspid* (three-pointed), by the older writers, *triglochine* (three-angled), its free margin forming three principal divisions: the *chordæ tendineæ* are the tendons, the *carneæ columnæ* the muscles of this valve; the tendinous cords converge from the point where they are attached to the valve, and several of them are inserted into a single fleshy column: when the latter contract, the tendinous cords are made tense; hence, as the fleshy columns contract during the ventricular systole, the valve is shortened, and prevented from being reversed.

Three principal divisions of the tricuspid valve are distinguished—an anterior, a right, and a posterior: the anterior division separates the auriculo-ventricular from the pulmonary orifice, and is supposed to prevent the blood from passing into the pulmonary artery during the dilatation of the ventricle; but (as was remarked by Senac long since) this can scarcely be necessary, because during the ventricular diastole the sigmoid valves close the arterial orifice; the second division of the valve lies to the right side; and the third, which constitutes the posterior division, lies behind. It would be more natural to consider this valve, like the mitral, as composed of two principal divisions: an anterior, which would include the anterior and right division, and a posterior, which would correspond to the posterior division: in describing the valve, however, it is convenient to consider it as composed of three curtains.

The anterior curtain is connected by its *chordæ tendineæ* principally with one large and long fleshy column, which has two roots: one of these roots springs from the anterior wall of the ventricle; the other, which runs transversely, is attached to the septum of the ventricles: this is the portion which Mr. W. King terms the “long moderator band.” The right curtain is connected by its fleshy columns to the parietes of the ventricle, not to the septum: two fleshy columns are attached in front; the others, which are much shorter, are attached to the posterior wall of the ventricle. The fleshy columns of the posterior division of the valve are much shorter than those of either of the others, and some of its tendinous cords are inserted without the intervention of any fleshy columns: they are all inserted into the septum.

A single slender tendinous cord, not proceeding from any fleshy column (which, however, is absent in some hearts), is attached by one extremity to the *anterior* division of this valve, sometimes near where the chordæ tendineæ join the fleshy column, more frequently to the fleshy column itself, either near its base or apex, from which it runs towards the right wall of the ventricle, into which it is inserted, or to the base of the fleshy columns of one of the other divisions of the valve. The action of this tendinous cord will be to prevent the anterior curtain of the valve from being applied to the orifice when the ventricle is much distended; and from its insertion into the yielding wall of the ventricle, the more the ventricular cavity is distended the more open will it keep this curtain of the valve; while when the ventricle is not over-distended, it will not interfere with the perfect action of the valve.

*Pulmonary Orifice.*—The orifice of the pulmonary artery is situated on a plane about three-quarters of an inch higher than the auriculo-ventricular orifice, to its left side and more anteriorly; the surface of the ventricle from which it arises, and below this point, is perfectly smooth. The pulmonary orifice lies pretty nearly on a line with the junction of the cartilages of the third ribs with the sternum, very little to the left of this bone; it has a circular shape, is smaller than the auriculo-ventricular orifice, and is provided with a valvular apparatus at the point where the artery joins the ventricle, formed of folds of the lining membrane, strengthened by fibrous tissue.

*Sigmoid Valves.*—The valves at the orifice of the pulmonary artery are three in number; they are analogous to the valves at the orifice of the aorta, but are thinner and more delicate. Morgagni gave them the name *sigmoid*, while those at the aortic orifice he named *semilunar*: these terms are, however, nearly indiscriminately used. The sigmoid valves have a crescentic shape as they lie against the walls of the artery: they are attached at the point where the pulmonary artery joins the ventricle, and this point or their base corresponds to a line across the inferior margin of the cartilage of the third ribs. One of these valves is anterior, another posterior or to the left side, and the third to the right side; each is attached by its convex margin, the concave or free margin being loose: their edges are a little thicker than the other parts, and in

the centre of each near the margin a very minute nodule is seated, though in some instances scarcely a trace of it is to be found.

The sigmoid valves, when the blood is passing out of the ventricle, lie against the walls of the pulmonary artery and present no impediment to its passage; when the ventricle ceases to contract, the blood in the artery above gets behind them, they fall down, close the orifice, and prevent regurgitation into the ventricle.

*Sinuses of the pulmonary Artery.*—Behind each sigmoid valve is a little dilatation of the vessel where its coats appear to be thinner: these are the sinuses of the pulmonary artery; their use evidently is to enable the blood to insinuate itself behind the valves when the ventricular systole ceases, by which they are pressed down, the orifice closed, and regurgitation into the ventricle prevented. These sinuses also allow a space for the sigmoid valves to lie back in during the ventricular systole, by which the outlet of the artery is rendered perfectly smooth and even, as the blood is passing from the ventricle into the pulmonary artery.

*Left Auricle.*—The left auricle occupies the upper, the posterior, and the left side of the base of the heart; it has an irregular square form, the transverse exceeding the vertical diameter; its parietes are thicker, and its cavity is smaller than that of the right auricle; its interior is smooth, the muscular fibres in it do not leave interspaces between them, and muscoli pectinati are fewer. It consists, like the right, of a sinus and an auricular appendix; the former receives the blood from the four pulmonary veins, and forms its principal bulk; it occupies the posterior part of the base of the heart, facing the bodies of the dorsal vertebræ, so that no part of it is visible when the pericardium is laid open. The auricular appendix (which is longer and narrower than that of the right side, and has its edge in general more deeply notched, or serrated), is situated at the lower and left side of the auricle, below the orifices of the left pulmonary veins; it comes forward at the left side of the base of the ventricle, where it lies to the left of the root of the pulmonary artery, and eventually a little in front of it; it is the only part of the left auricle which is visible when the pericardium is laid open, and will be found immediately under the cartilage of the third left rib. At the point where the auri-

cular appendix joins the sinus of the auricle, there is a narrowing or contraction of the diameter of the cavity: this is sometimes termed one of the orifices of the auricle, but incorrectly, because the sinus and appendix form but a single cavity.

In the interior of the left auricle five orifices are seen; the largest is the auriculo-ventricular orifice, the other four belong to the pulmonary veins; the latter open, one immediately above the other, at each side of the sinus, the two right at its posterior and right side, the two left at its posterior and left side. The two upper orifices are larger than the lower, and sometimes the two pulmonary veins of the left side open by a common orifice, sometimes there are three veins on the right side. The right wall of the auricle is formed by the septum auricularum; this, which is sometimes described as the base of the auricle, is convex towards the left side: in it we observe the depression which marks the site of the foramen ovale.

*Left Auriculo-ventricular Orifice.*—This orifice, which forms the communication between the auricle and ventricle, is situated in the floor of the auricle; it has an elliptic, or more correctly, a crescentic shape, the long diameter being nearly transverse; it is smaller than the corresponding orifice of the right side; its margin is smooth, and consists of dense, white, fibrous or tendinous tissue. The left auriculo-ventricular orifice is on a plane posterior to and to the left side of the tricuspid orifice, but nearly on the same level, as the heart lies *in situ*: it is situated behind the sternum, and upon a line with the junction of the cartilage of the fourth rib with that bone.

*Left Ventricle.*—The left, the aortic, or systemic ventricle, forms the left and posterior part of the body of the heart; it has a conical form, the base above where it joins the auricle, the apex below where it forms exclusively the apex of the heart; its parietes are convex externally, and concave internally, equally upon the side of the septum, as in its left and posterior wall, so that the septum seems to project into the cavity of the right ventricle. The walls of the left ventricle are strong, thick, and muscular, about three times the diameter of those of the right ventricle, as was first pointed out by Riolanus; they preserve, consequently, their convex form after the cavity of the ventricle is emptied, while the parietes of the right ventricle collapse under

similar circumstances. The left ventricle is longer than the right, but its cavity is smaller, and its carneæ columnæ are much thicker and stronger.

A small portion only of the left ventricle is seen when the pericardium is laid open; this is to the left of the sternum, extending from the cartilage of the third left rib, to the interspace between the fifth and sixth left ribs, near where the cartilage joins the body of these ribs. The appendix of the left auricle, which comes forward directly beneath the cartilage of the third left rib, surmounts the left ventricle here.

In the cavity of the left ventricle, as in that of the right, two parts are distinguished, an arterial and an auricular portion, separated from one another by the right or anterior division of the mitral valve. The former is the smooth surface leading to the aortic orifice; it is smaller than the corresponding portion of the right ventricle, and is bounded on the right side by the upper part of the septum of the ventricles, and posteriorly by the anterior or right curtain of the mitral valve. The auricular portion forms the larger part of the ventricular cavity: it communicates directly with the auricle on the same side.

The auriculo-ventricular and the arterial orifices lie much nearer each other on the left than on the right side of the heart, being only separated by the right or anterior curtain of the mitral valve. The aortic orifice is on a plane above, anterior to, and to the right of the auriculo-ventricular orifice: the right or anterior division of the mitral valve which separates them is supposed to prevent the blood from entering the aorta while the ventricle is filling; but as the semilunar valves are closed at this period, it can scarcely have this use.

*Mitral Valve.*—From the margin of the tendinous ring which surrounds the left auriculo-ventricular orifice, a valve composed of a double fold of the lining membrane, enclosing tendinous fibres, proceeds, the free surface of which hangs down into the ventricle; this valve is analogous in office to that of the right side, but is larger and stronger in all its parts: its chordæ tendineæ are stronger, and its carneæ columnæ are thicker.

This valve has more strictly two divisions than the tricuspid, and is properly termed *bicuspid* from this circumstance: one of these is anterior, the other posterior; the former being more to

the right side is sometimes termed the right division, the latter the left. Vesalius compared this valve to a bishop's mitre, "*quas mitræ episcopali non admodum inepte contuleris*," and the name *mitral* has been almost invariably applied to the valve since.

The anterior or right division of the mitral valve is the larger; it ascends higher than the other, reaching to the base of the posterior and right semilunar valves of the aorta: its curtain forms in a great measure the septum between the aortic and the auriculo-ventricular orifices, so that when we remove it the two orifices seem almost to constitute but one; indeed, it was formerly termed the valvular septum of Lieutaud from this circumstance. The *carneæ columnæ* of the mitral valve are all attached to the posterior wall of the ventricle: those of the anterior curtain arise from opposite sides of the posterior wall, each by two strong columns: the tendinous cords connected with them run towards one another, so that if continued along the curtain of the valve they would cross each other. The *carneæ columnæ* of the posterior division of this valve are three in number; they are shorter, broader, and less cylindrical than the fleshy columns of the anterior division. The strength of the *chordæ tendineæ* of this valve, notwithstanding their tenuity, is very remarkable: Senac tested it by isolating a single one, and attaching to it a four pound weight, which it supported for some time.

*Aortic Orifice.*—The aortic orifice, like the pulmonary, has a circular shape; it is situated at the upper and right corner of the base of the ventricle, behind the orifice of the pulmonary artery, and on a plane lower down. It lies anterior to, higher up, and to the right side of the auriculo-ventricular orifice, but very close to it; these two orifices being merely separated, as I have said, by the right or anterior curtain of the mitral valve.

*Semilunar Valves.*—The aortic orifice is provided with three valves, named semilunar by Morgagni, which are attached by their convex margin at the point where the aorta and left ventricle become continuous; their concave margin is free. They lie behind the sternum, towards the left side of this bone on a line with the space between the cartilages of the third and fourth ribs; their free edge corresponds (M. Gendrin observes) to the base of the pulmonary valves; a line drawn across the inferior margin of



the third rib corresponds to the free border of the aortic valves, and to the base of the valves of the pulmonary artery.

The semilunar valves of the aorta resemble the valves of the pulmonary artery but are thicker and stronger. They consist of duplicatures of the lining membrane, strengthened by tendinous bands; of these bands, one runs along the free margin of each valve, another along its base, and another, which is broader, runs upon each side of the corpus Arantii, and takes a semicircular course from this body to the attached margin of the valve. These tendinous bands have been recently described by M. Monneret as muscles: the action of one set of fibres (according to him) is to raise the valve, the other set are antagonists, and serve to depress them. But as the semilunar valves are elevated and depressed mechanically by the flux and reflux of the blood, it is not easy to perceive, (Magendie observes,) what purpose muscles placed between the membranous folds of these valves could serve.

*Corpora Arantii.*—Near the centre of the margin of the free edge of each valve a little fibro-cartilaginous body is seated, termed “corpora Arantii,” after Arantius, a pupil of Vesalius, who first accurately described them, though they had been noticed previously by Vidus Vidius; sometimes they are termed “corpora sesamoidea,” or “noduli Morgagni.” These little bodies are more distinct and larger in the semilunar valves of the aorta than in those of the pulmonary artery; their use is said to be to strengthen the central point where the valves meet, where the pressure is considerable, and the resistance least; as well as to fill up the little space which would be left in the centre when the valves fall down: but as these little bodies are not seated at the very margin of the valves, they could hardly effect the latter object, which, indeed, is unnecessary, as the valves slightly overlap one another in the healthy subject. The use of the corpora Arantii appears to me to be to serve as points of attachment for the tendinous bands already mentioned, by which the valves are strengthened; without some provision of this kind these valves would much more frequently become reversed, and permit regurgitation.

*Sinuses of the Aorta.*—Behind each semilunar valve the parietes of the artery are dilated, and a little pouch or sinus is formed, where the parietes of the artery are thinner than in other



parts; these are the sinuses of Morgagni, or the lesser sinuses of the heart. They are better developed in the aorta than in the pulmonary artery, and in old age than in young subjects. In these sinuses the orifices of the two coronary arteries are seen, and Dr. Chevers distinguishes those from which the right and left coronary arteries arise, by the names *right* and *left*; the third he calls the *intermediate* sinus. The disposition of the fibres of the sclerous coat of the aorta in these sinuses, by which the parietes are strengthened, and the attachment of the valve secured, has been well described by him. The use of these sinuses is similar to that of the same parts at the origin of the pulmonary artery; they allow a space for the blood to insinuate itself behind the valves when the ventricular systole ceases, by which they are pressed down and the orifice closed; and they afford a space for the semilunar valves to lie back in during the systole of the ventricle, by which the channel along which the blood passes is rendered perfectly smooth.

The aorta is connected to the left ventricle by the endocardium within, and by the serous and fibrous layers of the pericardium externally, which are continuous with one another in the heart and artery. When these are removed, the junction of the fibrous membrane of the artery with the muscular tissue of the heart is seen to be by three crescentic prolongations or festoons; each of which has its convex margin towards the ventricle, and between each is a small triangular interval, the base of which corresponds to the base of the ventricle.

The place where the aorta joins the ventricle is marked by a tendinous ring (*zona tendinosa*): the semilunar valves of the aorta are situated at this part, the convex margin of each being attached opposite to the convex margin of the crescentic prolongation above described, and each receiving tendinous fibres from this ring.

#### PERICARDIUM.

The pericardium belongs to the class of fibro-serous membranes and consists of two layers, an external fibrous and an internal serous. The fibrous layer is strong and resisting, though having but little thickness: it forms what is called the sac of the pericardium, and encloses the heart and the origin of the large vessels which come off from its base. The serous layer is thin and

delicate in comparison: it closely invests the heart, covers the origin of the large vessels, and is then reflected upon the internal surface of the fibrous layer, which it lines throughout. Thus, as is the case with other serous membranes, the heart, although invested by the pericardium, is not contained in its cavity.

The pericardial sac has a pyriform shape, the base below, the apex above; exactly the reverse of that of the heart. Its base is on a line with the upper part of the xyphoid cartilage; its apex a short distance above the origin of the large vessels, and generally on a line with the articulation of the cartilage of the second ribs with the sternum. I have, however, found it to extend, in a healthy subject, as high as the level of the articulation of the first ribs with the sternum. Its apex is higher upon each side of the aorta than immediately opposite to that vessel. The pericardial sac is wider in the centre than at its base, and here it extends more to the left side. Its widest part is on a line with the greatest transverse diameter of the heart.

*Capacity of the Pericardium.*—The capacity of the pericardial sac is somewhat greater than the volume of the heart; and, as its cavities are never all distended at the same moment, there is always abundant room for the apex and body of the organ to move freely in it. For the same reason, effusion of fluid to a moderate extent may take place into the cavity of the pericardium without interfering much with the heart's movements, provided it is effused slowly and gradually. On the other hand, even a moderate amount of fluid suddenly effused will be attended with formidable symptoms, because the fibrous nature of this membrane does not permit of its yielding suddenly. In chronic cases of disease, however, the pericardial sac is capable of becoming enormously dilated, as we shall afterwards see.

The actual capacity of the pericardium has been endeavoured to be determined by injecting fluid into and forcibly distending its sac. Dr. Sibson, in a recent number of the London Journal of Medicine, has given the following table of the results of some experiments made by him:

Boy, æt. 6, pericardium injected to distension, held . . .	6 oz.
"    9, . . . . .	6 oz.
"   13, . . . . .	about 6 oz.
Adult male . . . . .	15 oz.
Male, æt. 50 . . . . .	22 oz.
Adult female (heart enlarged) . . . . .	26 oz.

“From these and other observations,” Dr. Sibson says, “it may be inferred that in the adult, when the heart is healthy, the pericardium when fully distended, can contain from twelve to fifteen ounces of fluid.” “It is worthy of remark,” he adds, “that the right cavities of the heart in the adult male, when distended, hold the same quantity of fluid as the pericardium.”

*Fibrous layer of Pericardium.*—The fibrous layer of the pericardium is dense and strong, and is composed of tendinous fibres, some of which run vertically from the base towards the apex, others cross each other in various directions. Its tissue is semi-transparent, permitting the heart to be seen through it. It is very intimately united inferiorly to the central tendinous aponeurosis of the diaphragm: the fibres of each (as Lancisi observed) mix, and are confounded with one another; so that the pericardium seems as if it were a prolongation of this aponeurosis. It is also connected with the fleshy portion of the diaphragm on the left side, but the adhesion here is less intimate. Superiorly the fibrous layer of the pericardium is closely united to the trunks of the large vessels which come off from the base of the heart. It gives them sheaths; and, after accompanying them for a short distance, it becomes continuous with the thoracic fascia. The inferior cava alone receives no fibrous sheath from it. Anteriorly a small portion of its surface is only separated from the sternum by cellular tissue. Laterally, it is connected with the pleura upon each side, the phrenic nerve on the left side being interposed. Posteriorly, the pericardium, which is of small extent compared with its anterior surface, lies in front of the posterior mediastinum, being separated from the bodies of the dorsal vertebræ by the œsophagus and descending aorta.

The fibrous layer of the pericardium does not form a complete capsule for the heart; it is deficient inferiorly, where it is replaced by the central tendinous aponeurosis of the diaphragm; the reflected serous layer, which is attached to this part of the diaphragm, forming its capsule here. Owing to its density and strength the fibrous layer of the pericardium serves both to support and to limit the motions of the heart; while, it retains it *in situ*. From its firm attachments, above, below, and laterally, if as not unfrequently happens from disease, the opposed serous surfaces become adherent to one another, the motions of the heart must be, in a certain degree, interfered with.

*Serous Layer of Pericardium.*—The serous layer of the pericardium which invests the heart is thin and delicate, compared with the fibrous layer. It consists of two layers, a proper serous and a fibrous layer: the latter forms the capsule of the heart; the former, after covering the heart, ascends, from an inch and a half to two inches, upon the aorta, and upon the pulmonary artery as high as its bifurcation, where it is reflected upon the internal surface of the pericardial sac. Hence the greater portion of the ascending part of the arch of the aorta is said to be within the sac of the pericardium; and hence aneurism of this part of the vessel occasionally bursts into it. Where the serous layer lines the sac of the pericardium, it is very intimately adherent to it; where it covers the origin of the large vessels, it is much less so. Inferiorly, where the fibrous portion of the pericardial sac is deficient, the serous layer lines the central aponeurosis of the diaphragm.

*Cardiac fascia.*—The serous layer of the pericardium, which invests the heart, was always described as a single membranous layer, until Dr. Robert Lee\* demonstrated two layers in it, which are connected together by cellular tissue. The outermost of these layers is the proper serous coat: the inner layer, from its structure and function, and from the important office which it performs, Dr. Lee considers may be regarded as the fibrous membrane or fascia of the heart.

This expansion “is possessed of great strength and firmness. It is glistening, semitransparent, and resembles in all respects the aponeurotic expansions, or fasciæ covering muscles, in other parts of the body.” “It is much stronger over the ventricles than the auricles, and it adheres so firmly to the muscular substance underneath that its separation cannot be effected without tearing up some of the muscular fibres to which it is attached.” “From the inner surface of this fascia innumerable strong fibres pass to the blood-vessels, nerves, muscular fasciculi, and adipose matter, which accompany and surround all the blood-vessels and nerves; and they are interlaced together so as to form a peculiar stroma, if it may be so termed, of considerable thickness, between the fascia and all the various structures beneath, which it invests and binds together in the strongest possible manner. These fibres form a

\* Philosophical Transactions, 1848.

complete sheath around all the arteries, veins, and nerves, on the surface of the heart, and accompany them as they dip down between the muscular fasciculi, to which their branches are distributed throughout the entire walls of the heart, from its surface to the lining membrane."

"The cardiac fascia," Dr. Lee observes, "is obviously one of the principal causes of the firmness and strength of the central organ of the circulation, as it binds together into one mass, and gives support to the muscular fibres, like the fascia which invests other muscles." "The cardiac fascia is to the heart, I believe, what the external fibrous coat is to an artery; and it must have nearly the same effect in preventing dilatation and rupture of the ventricle during violent exertion." "The feeble serous covering of the heart can possess little influence, and add nothing to the strength of the parietes; and probably, but for the fascia now described, the heart would often yield in all directions, especially at the apex. In a physiological point of view, it therefore has appeared to me that this fascia of the heart is one of its most important strictures."

"In a pathological point of view," Dr. Lee observes, "the cardiac fascia is perhaps not less worthy of notice. Muscular structure, it is well known, is not liable to attacks either of common or of specific inflammation. It is impossible to avoid suspecting that rheumatic inflammation of the heart has for its principal seat this dense fibrous membrane lying between the serous and muscular coats of the heart, and that attacks of rheumatism of the heart do not commence primarily in the muscular structure."

The opposed serous surfaces of the pericardium are very smooth, and constantly lubricated: hence, in the normal condition of the parts, they glide over one another during the motions of the heart without producing sound. If, however, as is not unfrequently the result of disease, these surfaces become rough or uneven, increased friction must take place between them during the heart's motions, and sounds will be developed which become audible when the ear or stethoscope is applied to the parietes. These constitute the pericardial friction sounds, afterwards to be considered.

Owing to the intimate connexion of the pericardium inferiorly

with the diaphragm, it must follow the movements of the latter; and from its attachments above and below, its shape and state of tension must be somewhat different during inspiration and expiration; while if much fluid be effused into its sac, not only will the movements of the heart, the ascent of the diaphragm, and the expansion of the lungs be more or less interfered with, but the lungs will be pushed aside, a larger surface of the pericardium will come in contact with the parietes of the chest in front, and the region of the heart's superficial dulness on percussion will be increased in proportion. The effect of the forcible distension of the pericardium, in Dr. Sibson's experiments, was to make the central tendinous aponeurosis of the diaphragm convex towards the abdomen, and to lower this part about one inch.

*White Patch on Pericardium.*—Nothing is more common than to find upon the portion of pericardium investing the heart, a white patch of variable size and shape; this is the *milk spot* or *milk patch*, of the German writers, and it is so often found in hearts, which, in other respects, are perfectly healthy, that it has been regarded by some as a natural appearance. The anterior surface of the right ventricle is its most frequent seat; occasionally it is observed upon the surface of the left ventricle, or upon the auricles; in general only one exists, sometimes several are found upon the same heart; they have a white colour similar to that of healthy tendon; their shape is very variable, often somewhat circular or oval, occasionally more or less linear, and extending in the line of the coronary vessels. The size of these patches varies from a fourpenny piece to a crown, or even larger: when one only is present, it is usually larger than when several occur upon the same heart. They are much more common in the adult than in early life, but have been observed in the infant under three months; they are larger and better marked in advanced life, and they are more common upon the male than the female heart.

*Seat of the White Patches.*—Some difference of opinion exists among pathologists as to the exact seat of these opaque patches, some placing it upon the free surface of the pericardium, others upon its under surface, and others in the proper tissue of the membrane. Thus Baillie, Laennec, Louis, and Todd,\* state that

\* Cyclopædia of Anatomy and Physiology.

these opaque patches can easily be dissected or peeled off from the visceral layer of the pericardium, leaving this membrane entire. Corvisart, on the other hand, says, that these patches are seated upon the under surface of the membrane, and that they cannot be dissected off without bringing the pericardium with it. Dr. Hodgkin says, he has met with a few instances where they might be dissected off; but in by far the greater number of cases these patches depend on a deposit on the attached surfaces. Mr. T. W. King considers the actual seat of the deposit to be the proper tissue of the serous membrane. Mr. Paget\* says, these patches are “generally easily stripped off; but in no case after they are organized can they be separated from the subjacent tissue without dividing numerous connecting filaments, and leaving the surface from which they are removed flocculent and shreddy.”

*Nature and Causes of.*—A similar diversity of opinion exists among pathologists respecting the nature and cause of these opaque patches. By some they are referred to inflammation, and supposed to be always the result of partial pericarditis. Mr. Paget, who advocates this view exclusively, observes, “with these spots there almost constantly coincides some adhesion by organized lymph between adjacent parts of the pericardial membrane.” “The adhesions generally consist of slender threads passing across the furrow between the aorta and vena cava superior, or between the aorta and pulmonary artery at some little distance from their connexion with the heart.” In 40 cases noted by Mr. Paget, in which white patches were found upon the heart, 35 presented abnormal adhesions, or their remains.

If these white patches were always the result of inflammation, pericarditis, in a latent form, must evidently be a much more frequent disease than is commonly supposed; but as in the majority of cases the serous membrane at the part preserves its smooth and glistening appearance, the opacity can hardly be due to lymph deposited upon it; besides, adhesions between the visceral and the reflected layer of the pericardium, instead of being very rare in connexion with these patches, ought to be frequent, if they were always the result of the deposition of lymph.

Other pathologists regard these opaque patches as the effect of attrition between the surface of the heart and the parietes of the

\* Med. Chir. Trans. vol. xxiii.



thorax ; as they are found most frequently upon the anterior surface of the right ventricle. Dr. Hodgkin\* is of opinion that pressure is their cause, and that they are the result of a kind of inflammation originating in attrition and irritation. Mr. T. W. King† says, “the situation of these patches wherever they occur, implies to my mind a degree of attrition at the part more than belongs to the pericardium generally,” and he suggests the name “patches of attrition,” or “patches of distension” for them. It militates, however, against this theory, that these white patches are not unfrequent upon parts of the heart where attrition cannot take place. Other pathologists are of opinion that these patches are not to be regarded as a pathological phenomenon, because they are found on the most healthy hearts, and where the patient has never laboured under any affection referable to the heart ; and because similar opaque patches are found upon other viscera which are covered by serous membrane ; as the liver, intestines, &c.

M. Bizot, whose researches have tended to elucidate many doubtful points connected with the normal anatomy of the heart, has shown‡ that these white patches are of two descriptions, one probably consecutive to inflammation ; the other not a product of inflammation, but appearing to be in some way connected with the progress of age.

The first variety, or that which is the result of inflammation, is the rarest, it may occupy any part of the visceral layer of the pericardium, but its most usual situation is the anterior surface of the ventricles ; the opacity frequently also extends along the coronary veins, or adhesions exist between the base of the auricular appendages and adjoining parts. The shape of these patches is very irregular, the surface is often smooth, sometimes not ; they can usually be detached without difficulty from the serous membrane underneath.

The second variety, which is not the result of inflammation, consists of a thickened state of the pericardium itself, which undergoes slow transformation and loses its transparency ; it cannot, of course, be detached from the membrane. This is the most frequent variety, M. Bizot states that in 156 subjects he found it 45 times ; it is more frequent in the male than in the

\* Lect. on Morb. Anat. of Serous Membranes.

† Guy's Hosp. Reports.

‡ Mém. de la Soc. Med. d'Observation, Tome i.



male. The anterior surface of the right ventricle is its most frequent seat. In 45 instances, noted by M. Bizot, it occupied the centre of the anterior surface of the right ventricle in 20 ; in 8 it occupied, also, the anterior surface of the left ventricle ; in only it was found exclusively upon the left ventricle. The influence of age upon its development is also remarkable ; it was not found in the male under 17 years of age, or in the female under 22. After 40 years of age, in both sexes, these patches were not only better marked, but larger.

The following table, given by M. Bizot, shows its frequency at different ages, and in the two sexes :

MALE.			FEMALE.		
Age.	No. of subjects.	No. of examples	Age.	No. of subjects.	No. of examples
1 to 17	16	0	1 to 22	31	0
18 „ 39	24	8	23 „ 39	23	5
40 „ 79	32	23	40 „ 89	30	9

## ENDOCARDIUM.

The interior of the cavities of the heart is lined throughout by a transparent, delicate membrane named first by M. Bouilland *endocardium*. This membrane is very smooth, and highly polished, and is continuous with the lining membrane of the large vessels which open into the heart, as well as with the arteries and veins of the heart itself ; it lines every part of the interior of the cavities of both the auricles and ventricles, covers the chordæ tendineæ, and carneæ columnæ, and is reflected upon itself at the arterial and auriculo-ventricular orifices, to assist in forming the valves. It is thinner in the right cavities of the heart than in the left, and is said to be thickest in the left auricle ; it is so adherent to the tissue underneath that it can only be detached by the scalpel in small patches, the adhesion being closer about the valves than at any other part.

According to Luschka, “the endocardium is made up of the same structures which exist in the coats of the arteries, pressed together, and reduced to their extremest tenuity. The heart may thus be regarded as an expanded and modified vessel, with

muscular fibres disposed outside it." According to Rokitansky\* "the endocardium consists essentially, besides the epithelium of a longitudinal fibrous coat (Henle) under which there is a very considerable layer of elastic and cellular tissue, which is most distinct in the auricles, and on which rests the muscular substance of the heart. In the left side of the heart, more especially in the left auricle, a layer similar to that of the circular fibres of the arteries is occasionally found under the longitudinal fibrous coat." The epithelium and longitudinal fibrous coat, which constitute the true endocardium, are, like the inner coat of the arteries, non-vascular, but the subjacent tissue is abundantly supplied with vessels.

According to M. Bizot, the endocardium in early life has the same transparency, thickness, and consistence, in every part of the interior of the heart. Subsequently, a slight opacity begins to make its appearance in the vicinity of the semilunar valves of the aorta, and gradually increases, but is always most marked at the point at which it was first observed. As the membrane becomes opaque it appears also to become slightly thickened; but this is not due to the presence of a false membrane, it is a simple opacity, and appears to be analogous to the white patch upon the visceral layer of the pericardium. This appearance is only observed upon the left side of the heart. M. Bizot has never found it upon the right side; and age appears to have a marked influence upon its development; for it is very rare under puberty, and very frequent in old age.

#### MUSCULAR TISSUE OF THE HEART.

The muscular tissue which forms the principal bulk of the heart is situated between the endocardium on the one hand, and the pericardium on the other, and differs from the muscles of animal life in several respects; the latter are connected together by cellular tissue, and with the naked eye are seen to consist of bundles of fibres, while the former interlace and cross in different directions: under the microscope, also, other differences are observed,—as the granular appearance of the fibres, and the less distinctness of the transverse striæ. The substance of the heart

\* Manual of Pathological Anatomy, vol. 4.

contains a larger amount of muscular fibre for its size than any other part, and its tissue is compact, firm, and close ; for this reason the heart of animals when cooked, cuts uniformly smooth in every direction, and “eats short, not offering that elastic resistance which other muscles do during mastication.”

The use of the cellular tissue which connects together the fibres of the muscles of animal life, and which forms their sheaths, appears to be in a great measure to retain the muscular fibres within their proper sphere of action, and to strengthen them. In the heart cellular tissue was supposed to be less necessary, in consequence of the manner in which the muscular fibres themselves are arranged ; but Dr. Robert Lee \* has recently shown that the heart is provided with a fascia which binds together and gives support to the muscular fibres in the same way as the fascia which invests the muscles of animal life. This, which he terms the “*cardiac fascia*,” lies immediately under the true serous coat, and, after investing every part of the auricles and ventricles, it sends prolongations from its under surface, which surround the blood-vessels and nerves, forming sheaths for them, and accompanying their branches between the muscular fasciculi through the entire walls of the heart, from the surface to the lining membrane, investing and binding all the parts together in the strongest possible manner, and giving firmness and strength to the organ.

A vast deal of pains, time, and labour, have been expended by anatomists in endeavouring to unravel the course of the muscular fibres of the heart ; in many instances “they have succeeded (it has been observed) only in giving unintelligible descriptions.” Recently Mr. Searle has undertaken the task ; his account of the arrangement of the muscular fibres of the heart is contained in the “*Cyclopædia of Anatomy and Physiology* ;” his description would, however, be utterly unintelligible without the figures with which his paper is illustrated ; and, even with the assistance of these, the peculiar manner in which the bands, fasciculi, and layers, which enter into the formation of the heart, are arranged could scarcely be understood without quoting his description at length.

It will be sufficient here to observe that each ventricle has its own distinct set of fibres, which form its sac ; that another set of fibres surrounds, encloses, and unites together the two ventricles ;

\* Philosophical Transactions, 1849.

that in the left ventricle six layers of fibres have been distinguished; in the right, but three, which are arranged into a superficial, a middle, and an internal set; that the fibres are disposed in a spiral direction, "some winding round and round the ventricle," "some taking a larger sweep, and surrounding both ventricles;" that they interlace with one another, and that some are continuous with the *carneæ columnæ* in the interior of the ventricles.

The septum is described by Mr. Searle as consisting of three layers—a left, a middle, and a right; the two former belong properly to the left ventricle; the right layer exclusively to the right ventricle.

In the auricles the fibres are disposed in two layers: its fibres "arise chiefly from the tendinous margins of the *annulus venosus* and *annulus arteriosus*." The peculiar shape of the appendices depends upon the manner in which the fibres are arranged, as also the appearance known under the name of the *musculi pectinati*. The fibres of the right auricle are prolonged so as to form the outer part of the wall of the left auricle, which, as it receives additional bands of fibres, is necessarily thicker than the right auricle. The septum of the auricles receives fibres from three sources—superiorly, in its middle, and inferiorly.

The cause of the conical form of the ventricular portion of the heart is explained by Mr. Searle as follows:—"Along the central cavity of the left ventricle are placed the two *carneæ columnæ*, the length of which is equal to the lower three-fourths of the length of the axis of this cavity. The fibres of these two bodies radiate, and the radiated fibres wind round the axis closely upon them. By this radiation, instead of all the fibres passing longitudinally, which would have preserved these bodies in a state of equal thickness throughout their length, they are progressively parting with their fibres, retaining but a few, which by their longitudinal course, reach the apex; consequently these columns gradually diminish, becoming pyramidal, and form together an inverted cone; and as the fibres in well-formed hearts wind closely round these columns the entire ventricle gently assumes the form of a cone: and although the right ventricle is as it were appended to the left, yet it is not so connected to it as to destroy the conical form, but, on the contrary, in such a manner as to form a concave parabolic section of a cone, which adapts itself to the gentle cone of the left

ventricle. The two ventricles thus united assume the form of the more rapid cone of the heart."

#### NERVES AND BLOOD-VESSELS OF THE HEART.

The heart receives its nerves from both the ganglionic and cerebro-spinal system; the former are derived from the three cervical ganglions of the great sympathetic, and sometimes from the first dorsal ganglion; the latter are derived from the par vagum or pneumo-gastric nerve, and from its branch, the recurrent or inferior laryngeal nerve.

The three cardiac nerves upon each side, which are termed respectively the superior, the middle, and the inferior, come from the superior, the middle, and the inferior cervical ganglia of the great sympathetic. The superior cardiac nerve communicates with the middle and with the pneumo-gastric nerve; the middle communicates with both the superior and inferior, as well as with the pneumo-gastric and recurrent; the inferior communicates with the middle and with the recurrent nerves.

The cardiac ganglion receives the superior cardiac nerves on each side, and a branch from the pneumo-gastric, and sends branches to the cardiac plexuses. The great cardiac plexus is formed by the middle and inferior cardiac nerves from opposite sides; it receives branches also from the pneumo-gastric and recurrent: it lies behind the ascending portion of the arch of the aorta, in front of the trachea, near its bifurcation. From it lesser plexuses are formed, termed the anterior and posterior cardiac or coronary plexuses, which accompany the coronary vessels, supply the substance of the heart, and communicate with the pulmonary plexuses.

The experiments of Volkmann go to prove, that the movements of the heart are independent both of the brain and spinal cord; and that its movements "depend upon the ganglia and the nerve-fibres contained within itself; whilst centripetal fibres of these ganglia are found in the vagus, sympathetic, and in the spinal cord." It is to Dr. Robert Lee, however, that we are principally indebted for our knowledge of the ganglia and plexuses of the heart,—a subject which he has investigated with a patience and skill deserving of every praise. He has shown\* that every artery

\* Philosophical Transactions, 1849.

distributed throughout the walls of the heart, and every muscular fasciculus is supplied with nerves, upon which ganglia are formed. The number of these ganglia may be estimated by the fact, that as many as ninety are visible upon the nerves on the anterior surface of the heart.

The following conclusions, under this head, are given by Dr. Lee :

1. That the blood-vessels, and the muscular structure of the auricles and ventricles of the heart, are endowed with numerous ganglia and plexuses of nerves, which have not hitherto been described or represented in the works of anatomists.

2. That the nervous structures of the heart, which are distributed over its surface, to the apex and throughout its walls, to the lining membrane and columnæ carneæ, enlarge with the natural growth of the heart before birth, during childhood and youth, until the heart has attained its full size in the adult.

3. That the ganglia and nerves of the heart enlarge, like those of the gravid uterus, when the walls of the ventricles are affected with hypertrophy.

4. That the ganglia and nerves, which supply the left auricle and ventricle in the natural state, are more than double the size of the ganglia and nerves distributed to the right side of the heart.

*Blood-vessels of the Heart.*—The heart, although always more or less full of blood, cannot appropriate any of the fluid which passes through its cavities to its own nutrition; but, like every other organ, has peculiar vessels set apart to supply it: these are the two coronary arteries, which come off at the sinuses of Morgagni, immediately above the free margin of the semilunar valves. The principal division of the anterior or left coronary artery lies in the anterior longitudinal groove; its other branch runs round the base of the left ventricle, in the circular or auriculo-ventricular groove. The posterior or right coronary artery is lodged in the circular or auriculo-ventricular groove, and a large branch of it runs down the posterior longitudinal groove; they inosculate very freely with one another. The returning blood is principally conveyed by the great coronary vein which opens into the right auricle, between the Eustachian valve and the right auriculo-ventricular orifice. A valve formed by a semilunar fold of the lining membrane of the heart partially closes the

orifice, and prevents regurgitation. The grooves in which the coronary vessels run, mark the site of the septum of the ventricles as well as the line of separation between the auricles and ventricles; and owing to their position in these grooves, "they cannot," Dr. Wardrop observes, "suffer compression sufficient to interrupt their circulation, either from the auricular or ventricular contraction."

## CHAPTER II.

**DIMENSIONS OF THE HEALTHY HEART.—SIZE AND WEIGHT OF THE HEART.  
CAPACITY OF THE CAVITIES AND DIAMETER OF THE ORIFICES OF THE  
HEALTHY HEART.**

IN order to determine whether the heart is increased or diminished, or preserves its normal dimensions, it is absolutely necessary to have correct ideas respecting the size and weight of the heart; but as these vary with the age of the subject and with the sex of the individual, it is obvious that we can have no single standard for the adult heart.

Laennec laid it down that the closed hand, of the subject, was pretty nearly a mark of the size of the heart. This, however, is far from being the case: the heart of the infant at birth is larger than its closed hand, while the size of the hand varies according to the nature of the occupation of the person. It was supposed at one time that stature had considerable influence upon the size of the heart, and that this organ was always larger in tall than in short persons, in consequence of the greater distance to which the blood has to be transmitted. M. Bizot has, however, shown that stature has not the influence which was once supposed, and that whatever it exerts is rather in the opposite direction, the absolute size of the heart in both sexes being slightly less in tall persons: this, however, has been denied by M. Bouillaud. According to M. Bizot, the breadth across the shoulders has a greater influence than stature; the mean size of the heart and the width of the shoulders in both sexes being in a pretty regular ratio.

That the heart increases in size with the age of the individual, that in advanced life it is larger than in the adult, and that it is larger in proportion in the male than the female at every age, has been satisfactorily proved by M. Bizot. The results of accurate measurements of the heart in 156 individuals of all ages, given



by him, show that this organ regularly and progressively increases in all its dimensions—length, breadth, and thickness—up to the latest period of life; the increase being more rapid before twenty-nine years of age than after that period: and that the heart of the female at every period of life is smaller than that of the male.

The late Dr. Clendinning, whose researches were carried on about the same time, but without any knowledge of M. Bizot's labours, arrived at very nearly similar conclusions: his inquiries were particularly directed to determine the absolute weight of the heart in health and disease, as well as its relative weight to the entire body. Dr. Ranking, likewise, from the examination of a number of healthy hearts, and M. Neucourt from researches carried on at the Salpêtrière, in the service of MM. Valleix and Beau, have been able to corroborate some of M. Bizot's conclusions. M. Neucourt's examinations were limited to females in advanced life.

*Length of the Heart.*—The length of the healthy heart in the adult, according to Meckel, measured from the centre of the auricles, is between five and six inches, four of which are for the ventricular, and one and a-half for the auricular portion. Senac considered from three and a-half to four inches to be the average length of the ventricular portion of the heart. According to M. Bouillaud, the mean length of the heart, from the base of the left ventricle or from the aortic orifice to the apex of the organ, in nine subjects was three inches seven lines and three-quarters. Dr. Ranking\* from an accurate examination of thirty-two healthy hearts (fifteen male and seventeen female) gives as the mean length of the heart, measured from the point where the aorta emerges to the apex of the organ, four inches and one-third for the male, and three inches and a-half for the female. In my examinations, the length of the heart, measured from the summit of the appendix of the right auricle to the apex of the left ventricle, ranged from four inches and a-quarter to five inches and a-half. I look upon five inches and a-half to be the full length of the healthy adult heart.

*Breadth of the Heart.*—The breadth of the heart across the base of the ventricles Meckel considers to be three inches; Senac, between two, and two inches and a-half; M. Bouillaud states that

\* London Medical Gazette.

the average in eight hearts was three inches seven and a-half lines, and the mean circumference at the base of the ventricles eight inches nine lines and three-sevenths. Dr. Ranking gives the mean circumference of the base of the heart in the male as slightly above nine inches and a-half; in the female, about eight inches and a-quarter. In the examinations which I have made, I have found the breadth of the heart across the ventricles to range between three inches and a-quarter and four inches.

The following table, given by M. Bizot, shows the progressive increase in length and breadth of the heart from infancy to old age, as well as the comparative size of the heart in the male and female at different ages. The measurements are given in lines; the length is measured from the base of the ventricular portion to the apex; the width, at the part where the auricles and ventricles join one another.

				FEMALES.			
Age.	Number of subjects.	Length.	Breadth.	Age.	Number of subjects.	Length.	Breadth.
1 to 4	7	22 $\frac{1}{2}$	27	...	8	22 $\frac{1}{2}$	25 $\frac{1}{2}$
5 to 9	3	31 $\frac{1}{2}$	33	...	10	26 $\frac{1}{2}$	29
10 to 15	3	34	37	..	5	29 $\frac{1}{2}$	31 $\frac{1}{2}$
16 to 29	18	42 $\frac{5}{8}$	45 $\frac{1}{8}$	...	14	38 $\frac{1}{2}$	42 $\frac{3}{4}$
30 to 49	23	43 $\frac{5}{8}$	47 $\frac{1}{8}$	...	27	41 $\frac{3}{4}$	44 $\frac{1}{4}$
50 to 79	19	45 $\frac{1}{8}$	52 $\frac{1}{8}$	50 to 89	19	42 $\frac{3}{8}$	46 $\frac{1}{8}$

*Conclusions respecting the size of the Heart.*—From the preceding details, it appears that—

1. The heart in both sexes gradually and progressively increases in size from infancy to old age; the increase being more rapid up to twenty-nine years of age than after that period.

2. The heart of the male is larger in all its dimensions, at every period of life, than that of the female; and the ratio is nearly the same at all ages except infancy.

3. The breadth across the base of the ventricles exceeds the length of the ventricles at every age, and in both sexes.

4. The mean length of the ventricular portion of the healthy heart of the male, at the period of life when this organ attains its maximum development, is slightly under three inches ten lines; in the female, under three inches seven lines.

5. The mean breadth of the ventricular portion of the healthy heart of the male in advanced life is below four inches five lines ; in the female, below three inches eleven lines.

6. In persons of elevated stature the absolute size of the heart is not only not greater than in individuals of medium height, but, from M. Bizot's researches, appears to be rather less.

7. The capacity of the thorax, indicated by the width of the shoulders, has a greater influence upon the size of the heart than stature ; and this organ is larger, as a general rule, where the shoulders are wide apart than in individuals with narrow shoulders.

#### WEIGHT OF THE HEART.

Lancisi was the first to endeavour to determine the normal condition of the heart by weighing it ; but until within the last few years we had no positive information respecting either the absolute weight of the healthy heart, or the ratio which the weight of the heart bears to that of the entire body ; and it is to the researches of the late Dr. Clendinning that we are principally indebted for statistics under these heads. He has shown\* that the absolute weight of the healthy heart varies with the age and sex of the subject ; that in the female the weight is always below that of the male ; and that in the male the weight increases gradually with the years as life advances ; while in the female, although the absolute weight is rather less in old age than about fifty, the relative weight to the entire body increases as life advances after that period.

*Absolute weight of the Heart.*—Senac considered the weight of the healthy heart of the adult to be between eight and ten ounces. Meckel lays down ten ounces as its average weight ; Lobstein estimated it at between nine and ten ounces ; Cruveilhier at between six and seven ounces ; and Sanson at eight ounces and a-half. Bouillaud says we may fix the mean weight of the heart in the adult between twenty-five and sixty years of age, at between eight and nine ounces ; in thirteen cases given by him, the average was eight ounces three drachms, the maximum being eleven ounces, and the minimum being six ounces two drachms : the former, he observes, belonged to a subject of colossal stature, and of strong constitution : the latter to a subject of only sixteen years of age.

\* Med. Chir. Transactions.

The following table, given by Dr. Clendinning,\* shows the net average weight of the healthy heart, at different ages, in the two sexes. Forty-eight of the subjects were males, and ninety-eight females; none are included in the table in which the weight of the heart reached eleven ounces avoirdupois:—

Age.			Male.		Female.
15 to 30	.	.	8½ oz.	.	8½ oz.
30 to 50	.	.	8½	.	8½
50 to 70	.	.	9½	nearly	8
70 to 100	.	.	9½	.	8

*Ratio of weight of Heart to the Body.*—The ratio which the weight of the heart bears to the entire body is different at different ages, and in the two sexes. According to Meckel, in the infant soon after birth the ratio is as one to one hundred and twenty, while in the adult it is as one to two hundred. But, as Dr. Clendinning remarks, Meckel has not given particular observations, and possibly did not pay sufficient attention to differences of age, sex, and disease. The following table, given by Dr. Clendinning, shows the ratio which the weight of the heart bears to the entire body at different ages, and in the two sexes. This table includes twenty-three subjects under puberty, most of whom were under five years of age: forty-two males and fifty-eight females above puberty:—

Age.			Male.	Female.
Under puberty	.	.	1 to 146	1 to 153
15 to 30	.	.	1 to 164	1 to 169
30 to 50	.	.	1 to 150	1 to 161
50 to 70	.	.	1 to 161	1 to 187
70 and upwards	.	.	1 to 155	1 to 121

“The absolute weight of the healthy heart may therefore,” Dr. Clendinning observes,\* “be assumed to average for the whole of life above puberty, about nine ounces for the male, and eight ounces, or a little more, for the female; and to bear after death to the entire body the ratio of about one to one hundred and sixty for the male, and one to one hundred and fifty for the female. So that a male adult heart considerably exceeding the 160th part of the whole subject, might, for a person of the working classes, and

\* London Medical Gazette, Vol. xxii.

ordinary fatness, but of much muscularity, be held to be normal ; while a heart of like absolute dimensions, but occurring in a subject of average stature and muscularity, would be justly considered hypertrophous, although, owing to general obesity or œdema, it could not exceed, or should even fall short of, the 160th part of the weight of the person. And this observation applies, *mutatis mutandis*, to the female ; in whom, I think, the extremes of obesity and leanness more frequently occur than in the male." The female stature and muscular development vary considerably less than the male, and the limits of normal nutrition, and the volume of the heart, are consequently narrower ; so that any male heart greatly exceeding eight ounces in weight or volume may *ipso facto* be suspected of hypertrophy, whatever may be its parent anatomical condition ; and but few instances indeed, if any, will be met with, I apprehend, of female hearts exceeding nine ounces in volume, or nine and a-half in weight, in the persons of individuals of ordinary stature and conformation, that had been quite free from pectoral disease during their lives."

*Comparative weight of parts of the Heart.*—We possess but few statistics bearing upon the comparative weight of the several parts of the heart one to another. According to Valentin, the weight of the left ventricle is double that of the right ; and he calculates from this that the force exerted by the right ventricle in its systole is only half that of the left ; allowing two-thirds of the force of the septum to the left ventricle and one-third to the right. Ludwig states as the result of weighing the muscular substance of the ventricles, in both a moist and dry state, that the tissue of the left ventricle contains much less water than the right ; consequently it has a much greater relative weight when dried.

#### *Conclusions respecting the weight of the Heart :—*

1. The absolute weight of the heart increases with the years, as life advances, in a very perceptible manner in the male. In the female there is no increase in its absolute weight after fifty years of age.

2. The absolute weight of the heart is greater in the male than in the female at every age.

3. The average absolute weight of the healthy adult heart

may be set down as nine ounces for the male, and eight ounces, or a little more, for the female.

4. The ratio which the weight of the heart bears to the entire body is different at different ages, and in the two sexes.

5. In infancy, in both sexes, the ratio of the weight of the heart to the entire body is greater than in the adult. In advanced life it is greater than at the middle periods.

6. In the female in advanced life the ratio of the weight of the heart to the entire body is much greater than in the male at the same period of life.

7. In estimating the weight of the heart, in doubtful cases, the relative as well as the absolute weight of the organ ought to be taken.

#### THICKNESS OF THE WALLS OF THE HEART.

The parietes of the several cavities of the heart have not the same thickness: as a general rule, the walls of the left chambers are thicker than those of the right, and the parietes of the ventricles much exceed those of the auricles; the left ventricle has much thicker walls than the right, and the walls of the left auricle are a little thicker than those of the right.

*Left Ventricle.*—According to M. Bouillaud,\* the average thickness of the walls of the left ventricle, near its base, in ten subjects, was six lines and a-half; we may consider (he adds) seven lines as the average thickness of the parietes of this ventricle. According to Dr. Ranking,† the mean thickness of the walls of the left ventricle in the male, at a point about an inch distant from the origin of the aorta, is 27-48ths of an inch; in the female 23-48ths of an inch. M. Bizot's researches‡ prove that the parietes of the left ventricle, in both sexes, go on increasing in thickness from youth to advanced age; and that the thickest part of its walls is at the middle, next at the base, and that the thinnest part is at the apex.

The accompanying table, given by him, shows the mean thickness (in lines) of the parietes of the left ventricle at these three points, in the male and female, at different ages.

\* *Traité des Maladies du Cœur*, 2nd edition.

† *London Medical Gazette*.

‡ *Mém. de la Soc. Med. d'Observation*, tome i.

LEFT VENTRICLE.

MALE.				FEMALE.			
Age.	Base.	Middle.	Apex.	Age.	Base.	Middle.	Apex.
16 to 29	4 2-9	3 7-9	3 4-9	16 to 29	4 2-7	4 7-13	3 5-28
30 to 49	4 17-46	5 1-11	3 13-23	30 to 49	4 1-9	3 27-54	3 6-27
50 to 79	4 37-38	5 29-38	4 1-29	50 to 89	4 1-2	5	3 3-4
Mean average between 16 and 79	4 65-122	5 19-122	3 95-122	Mean average between 16 and 89	4 3-8	4 4-5	3 13-30

*Right Ventricle.*—According to M. Bouillaud, the average thickness of the walls of the right ventricle in ten subjects was two lines three-fifths: we may therefore, he says, consider two lines and a-half as the average thickness of the parietes of the right ventricle. According to Dr. Ranking, the mean thickness of the walls of the right ventricle in the male, at a point about an inch distant from the origin of the pulmonary artery, is 8-48ths of an inch; in the female 6-48ths of an inch. According to M. Bizot, the thickest part of the parietes of the right ventricle is not at the same point as on the left side, but is close to the base of the heart; and the parietes of this ventricle remain nearly stationary at the different periods of life, being, however, a little thicker in advanced life than at an earlier period.

The following table, given by M. Bizot, shows the mean thickness (in lines) of the parietes of the right ventricle at three distinct points, in the male and female at different ages.

RIGHT VENTRICLE.

MALE.				FEMALE.			
Age.	Base.	Middle.	Apex.	Age.	Base.	Middle.	Apex.
16 to 29	1 27-38	1 8-19	1 1-19	16 to 29	1 4-7	1 2-7	25-28
30 to 49	1 39-46	1 7-23	45-46	30 to 49	1 19-22	1 13-54	25-27
50 to 79	2 1-19	1 53-168	81-84	50 to 79	1 1-4	1 1-4	1
Mean average between 16 and 79	1 113-122	1 99-244	1 2-61	Mean average between 15 and 59	1 2-3	1 7-24	673-720

*Septum Ventriculorum.*—According to Dr. Ranking, the mean thickness of the septum of the ventricles, taken at its centre, in fifteen males between 26 and 65 years of age, was 22-48ths of an inch. In seventeen females, between the age of 18 and 62, 14-48ths of an inch. M. Bizot's researches show that the thickness of the septum of the ventricles goes on increasing from infancy to an advanced period of life; and that it is thicker in the male than in the female. The following table, given by him, shows the average thickness (in lines) of this part at its centre, in the male and female at different ages.

Age.	Male.	Female.
16 to 29	4 17-18	4 11-14
30 to 49	4 21-23	4 11-27
50 to 79	5 1-3	5 3-19

*Auricular Walls.*—The walls of the left auricle exceed those of the right in thickness; but statistics are wanting under this head. M. Bouillaud is almost the only writer who has given measurements. According to him, the average thickness of the parietes of the left auricle in four subjects was one line and a-half; of the right auricle one line.

*Conclusions respecting the thickness of the Parietes of the Heart.*—From the preceding details it appears that—

1. The parietes of the left ventricle are thicker in the male than in the female heart at every age.
2. The parietes of the left ventricle and of the septum ventriculorum increase in thickness as age advances in both sexes.
3. The parietes of the right ventricle increase also in thickness as age advances in both sexes, but in a much less ratio.
4. The thickest part of the parietes of the left ventricle is at the centre, next at the base, and it is thinnest at the apex.
5. The thickest part of the parietes of the right ventricle is at the base.
6. The thickest part of the septum ventriculorum is at its centre.
7. The mean thickness of the parietes of the left ventricle, or of the septum, in the healthy heart of the male in advanced life,



when the walls of the heart attain their maximum development viz. between 50 and 79 years of age), and at the thickest part, is under six lines.

8. The mean thickness of the parietes of the left ventricle in the female between 50 and 89 years of age, is five lines.

9. The mean thickness of the parietes of the right ventricle in the healthy heart of the male, between 50 and 79 years of age, and at its thickest part, is a fraction above two lines; in the female, one line and a-quarter.

10. The mean thickness of the parietes of the left ventricle is, therefore, in both sexes, nearly three times as great as that of the right ventricle, as was long since laid down by Riolanus.

#### CAPACITY OF THE CAVITIES.

The capacity of the several cavities of the healthy heart is not the same: the auricles exceed the ventricles in this respect; the right auricle likewise exceeds the left, and the capacity of the right ventricle is greater than that of the left. The capacity of both ventricles increases gradually as life advances in both sexes; the increase is more rapid in youth; after fifty years of age it is less marked, although it still goes on; so that, both in infancy and old age, the ratio between the capacity of both ventricles is the same.

That the capacity of the right ventricle is greater than that of the left was suspected long before it was actually proved. Senac says repeated examinations have convinced him that the capacity of the right ventricle exceeds that of the left; but that the difference is almost insensible in some subjects. M. Bouillaud repeats Senac's words: he says—"the medium capacity of the right ventricle exceeds that of the left; but the difference is really very slight." This position has, however, been denied by some writers, and the apparently increased capacity of the right ventricle has been supposed to depend upon the accumulation of blood in the right cavities of the heart after death. "There is much reason to believe," Dr. A. Thomson observes,\* "that the greater capacity of the right auricle and ventricle depends in part upon the accumulation of blood which generally takes place in most kinds of slow death in the pulmonary arteries; and in part also

\* Cyclopædia of Anatomy and Physiology.

upon the greater thinness, and consequent distensibility, of the right ventricle. In men dying suddenly, and in animals killed purposely, in which the pulmonary artery is opened so as to allow of the free egress of the blood from the right side of the heart, the capacity of this ventricle is not greater than that of the left, and the proportions of the capacity of the two sides of the heart usually found after slow death are sometimes reversed when a ligature is placed on the aorta, and the pulmonary artery is opened." But, as was long since remarked by Senac, "since in the foetus the capacity of the right ventricle exceeds that of the left, it would be strange if in the adult they should have the same capacity;" while M. Bizot's researches have proved that the right ventricle exceeds the left in capacity at every age, that this is the normal condition of the parts, and that it is not at all owing to the accumulation of blood during the last moments of life.

The actual capacity of each of the ventricles has been endeavoured to be determined by filling their cavities with fluid or with injection; but, as the parietes of the right ventricle are much more yielding than those of the left, this method is calculated to lead into error; indeed, Senac says he has introduced a pound and a-half of injection into the cavities of the heart without using much force. According to him, each ventricle holds about two ounces of fluid; but the excess over this is very variable, the right sometimes holding from one to four drachms more. Hales estimated the capacity of the left ventricle at one fluid ounce and a-half, and that of the right at two fluid ounces. Each ventricle, on an average, it is generally considered, will contain a hen's egg, and, when moderately distended, is capable of holding from an ounce and a half to two fluid ounces, or a little more.

M. Bizot determined the capacity of the ventricles by laying open their cavities, and measuring accurately the internal surface from above downwards, and transversely. From his researches it appears that both ventricles are wider than long in both sexes, and at every age; that the length, but particularly the breadth of the right ventricle, exceeds that of the left at all ages; that the capacity of the ventricles is greater in the male than the female; and that the relative capacity of the two ventricles is pretty nearly the same in advanced life and in infancy.

The capacity of the right auricle exceeds that of the left: the

proportions established in the “Mémoires de l'Académie” between them are as twenty-four to thirteen; according to Santorini, as five to three; but the inequality is seldom so marked in the perfectly healthy heart. According to Dr. Sibson,\* the capacity of the right auricle is about the same as that of the right ventricle; while the capacity of the left auricle is less than that of the left ventricle. He, however, estimates the capacity of both ventricles as the same.

*Conclusions respecting the capacity of the Cavities of the Heart :—*

1. The capacity of both ventricles increases gradually as life advances in both sexes, the increase being more rapid in youth.

2. The ratio between the capacity of each ventricle is pretty nearly the same at all ages.

3. The capacity of both ventricles is greater in the male than in the female.

4. The capacity of the right ventricle much exceeds that of the left at all ages, and in both sexes.

5. The right ventricle holds, when moderately distended, about two fluid-ounces; the left ventricle about one ounce and a-half.

6. The capacity of the right auricle exceeds that of the left; and the capacity of the auricles is greater than that of the corresponding ventricles; but further statistics are required under this head.

DIAMETER OF THE ORIFICES.

The size, as well as the shape, of the arterial and auriculo-ventricular orifices is different: the former have a circular, the latter rather an elliptic form; the left auriculo-ventricular orifice has more of a crescentic shape than the right. The orifices of the right side of the heart exceed in size those of the left: this, however, applies more to the auriculo-ventricular than to the arterial orifices, as in advanced life the aortic orifice rather exceeds the pulmonary in size. M. Bizot's researches likewise show that the diameter of all the orifices of the heart is greater in the male than in the female, and that their size progressively increases as life advances in both sexes from puberty to old age.

\* Trans. of Prov. Association, vol. xii.

In order to determine the diameter of the orifices of the heart, the most convenient method is to lay open the part by an incision through it, and take its circumference, from which the diameter is readily calculated. When the orifice is diseased, or if we wish to preserve the preparation, we must be content with measuring the diameter by means of a pair of compasses.

*Right auriculo-ventricular Orifice.*—According to Cruveilhier, the long diameter of this orifice is from sixteen to eighteen lines ; its short diameter twelve lines. M. Bouillaud found the average circumference of this orifice in three healthy hearts to be three inches ten lines ; the maximum being four inches, the minimum three inches nine lines. According to Dr. Ranking, the mean circumference of this orifice in fifteen males was 4 inches 35-48ths ; in seventeen females, 4 inches 8-48ths. In the measurements which I have made, I have found the long diameter of this orifice to range between fourteen and sixteen lines and a-half.

The following table, given by M. Bizot, shows the mean circumference (in lines) of this orifice at different ages in the male and female.

RIGHT AURICULO-VENTRICULAR ORIFICE.

MALE.		FEMALE.	
Age.	Lines.	Age.	Lines.
16 to 29	50 9-19	16 to 29	37 12-13
30 to 49	54 5-23	30 to 49	47 4-27
50 to 79	57½	50 to 89	49 13-19
Mean average between 16 and 79	54 12-61	Mean average between 16 and 89	48½

*Left auriculo-ventricular Orifice.*—According to Cruveilhier, the long diameter of this orifice is from thirteen to fourteen lines ; its short diameter from nine to ten lines. M. Bouillaud states that the average circumference in three hearts examined by him, was 3 inches 6½ lines ; the maximum being 3 inches 10 lines, the minimum 3 inches 3 lines. According to Dr. Ranking, the mean circumference of this orifice in the male is 3 inches 45-48ths ;

in the female, 3 inches 22-48ths. In the examinations which I have made, the long diameter of this orifice, measured from the auricle, ranges between ten and twelve lines.

M. Bizot has given the following table of the mean circumference (in lines) of this orifice in the two sexes at different ages :

LEFT AURICULO-VENTRICULAR ORIFICE.

MALE.		FEMALE.	
Age.	Lines.	Age.	Lines.
16 to 29	41	16 to 29	38
30 to 49	48 9-22	30 to 49	40 17-26
50 to 79	48 13-19	50 to 89	44 4-19
Mean average between		Mean average between	
16 and 79	45 17-30	16 and 89	41½

*Aortic Orifice.*—According to M. Bouillaud, the average circumference of the aortic orifice in four healthy hearts was 2 inches 5½ lines; the maximum being 2 inches 8 lines; the minimum, 2 inches 4 lines. According to Dr. Ranking, the mean circumference of this orifice, on a line with the insertion of the semilunar valves, in fifteen males between twenty-six and sixty-five years of age, was nearly 2¾ inches; in seventeen females, between eighteen and sixty-two years of age, 2 inches 22-48ths. According to Dr. Chevers, the circumference of the aortic orifice, immediately below the semilunar valves, is 36½ lines; above them, 34 lines. In the examinations which I have made I have found the diameter of this orifice to range between ten and twelve lines. The accompanying table, given by M. Bizot, shows the mean circumference (in lines) of the aortic orifice, at the free border of the semilunar valves, in the male and female, at different ages.

AORTIC ORIFICE.

MALE.		FEMALE.	
Age.	Lines.	Age.	Lines.
16 to 29	26 10-19	16 to 29	24 3-9
30 to 49	30 20-23	30 to 49	28 3-27
50 to 79	36	50 to 89	32 5-6
Mean average between		Mean average between	
16 and 79	31 15-61	16 and 89	28 4-5

*Pulmonary Orifice.*—M. Bouillaud found the mean circumference of the pulmonary orifice in four healthy hearts to be 2 inches 7 $\frac{3}{4}$  lines; the maximum being 2 inches 10 lines; the minimum, 2 inches 6 lines. According to Dr. Ranking, the mean circumference of the pulmonary orifice in the male is 2 inches 34-48ths; in the female, 2 inches 24-48ths.

The following table, given by M. Bizot, shows the mean circumference (in lines) of this orifice, at the free border of the sigmoid valves.

PULMONARY ORIFICE.

MALE.		FEMALE.	
Age.	Lines.	Age.	Lines.
16 to 29	29 2-19	16 to 29	28 3-14
30 to 49	31 12-23	30 to 49	29 $\frac{1}{2}$
50 to 79	35	50 to 89	32 17-36
Mean average between 16 and 79	32 21-61	Mean average between 16 and 89	30 7-60

*Conclusions respecting the diameter of the Orifices of the Heart.*—From the preceding details it appears that,

1. The diameter of all the orifices of the heart is greater in the male than the female, at every age.
2. The diameter of all the orifices of the heart increases gradually from birth to old age in both sexes.
3. The auriculo-ventricular orifices exceed the arterial orifices in size at every age, and in both sexes.
4. The right auriculo-ventricular orifice exceeds the left in size in both sexes, and at every period of life, except in the female between the ages of sixteen and twenty-nine, when, according to M. Bizot, there is a very slight excess of the left over the right.
5. The pulmonary orifice exceeds the aortic orifice in size in both sexes, up to the age of fifty; after this period the aortic orifice slightly exceeds the pulmonary orifice.
6. The mean circumference of the right auriculo-ventricular orifice, in the healthy heart of the male, in advanced life, when the

orifices of the heart attain their maximum size—viz. between fifty and seventy-nine years of age—is, omitting fractions of lines, 4 inches 9 lines; of the left auriculo-ventricular orifice, four inches.

7. The mean circumference of the right auriculo-ventricular orifice in the female, between fifty and eighty-nine years of age, is (omitting fractions of lines) 4 inches 1 line; of the left, 3 inches 8 lines.

8. The mean circumference of the pulmonary orifice in the male in advanced life, is 2 inches 11 lines; of the aortic orifice, 3 inches.

9. The mean circumference of the pulmonary orifice in the female, between fifty and eighty-nine years of age, is about 2 inches 8 lines; of the aortic orifice, 2 inches 8 lines.

#### RELATIVE POSITION OF THE SEVERAL PARTS OF THE HEART TO THE PARIETES OF THE CHEST.

A knowledge of the exact position of the several parts of the heart, particularly of its valves and orifices, and of their relation to fixed points on the surface of the chest, is of considerable importance in the diagnosis of the diseases of this organ, as it enables us to determine the size of the heart in the living subject, to ascertain whether it is increased or diminished in any of its diameters, or whether it is displaced, and the amount of the displacement; and by it we can often determine which of the valves or orifices is diseased, as well as the nature of the morbid change.

The apex and body of the heart being capable of some change of position, according to the posture of the patient, and according to the motions of the diaphragm; and the base of the organ, from which the large vessels proceed, being more fixed, the latter is the most convenient point from which to trace the outline of the heart, or to determine what parts correspond to certain fixed points upon the surface of the chest.

The sternum in front, with the cartilages of the true ribs laterally, form in a great measure, the anterior boundaries of the heart; a small portion only of this organ near its apex extends on the left side beyond the cartilages of the ribs. The base of the ventricular portion of the heart is from three to three and a-half inches below the clavicle, and on a line with the junction of the cartilage of the third left rib with the sternum. The impulse of

the apex of the organ is felt between the fifth and sixth left ribs, near where the body of these ribs joins the cartilage. A line, therefore, drawn between these two points gives the length of the ventricular portion of the healthy heart. In addition, the nipple in the male constitutes a useful guide: it is situated immediately opposite the lower edge of the fourth rib, a little more than an inch to the left of the junction of this rib with its cartilage, and as the edge of the left ventricle reaches the nipple on that side, and as the right ventricle extends a little to the right of the sternum, a line drawn between these two points gives its transverse diameter.

The greater part of the *right ventricle* lies behind the sternum, at its upper part it extends slightly to the right of this bone, its apex is to the left of the sternum. It is overlapped by both the right and left lung; below where the margins of the lungs separate from one another a small triangular space is uncovered by lung.

The *left ventricle* is covered by the left lung; all its anterior surface is to the left of the sternum; its left margin reaches to the nipple on the left side.

The *right auricle* lies to the right of the sternum, and is entirely covered by the right lung; its appendix lies behind the cartilage of the third right rib; its tip rests against the right side of the ascending portion of the arch of the aorta, and is on a line with the pulmonary valves.

The *left auricle* is entirely covered by the left lung, its appendix is the only portion seen when the pericardium is laid open; it lies behind the cartilage of the third left rib, close to the sternum, resting against the left side of the commencement of the pulmonary artery.

The base of the heart—that is, of the auricular portion—is on a line with the interval between the junction of the second and third ribs with the sternum, the greater portion of it being under the sternum.

The base of the ventricular portion of the heart on the left side rises as high as a line drawn across the junction of the cartilage of the third left rib with the sternum; that is, about three inches below the clavicle on that side. On the right side, owing to the oblique position of the heart, the base of the right ventricle corresponds to a line across the upper margin of the junction of the cartilage of the fourth right rib with the sternum.



The apex of the left ventricle is a little below the fifth left rib, to the left of the junction of this rib with its cartilage, and on a line with the articulation of the xyphoid cartilage with the sternum.

## RELATIVE POSITION OF THE ORIFICES.

*The right auriculo-ventricular orifice* lies behind the centre of the sternum, on a line with the lower margin of the articulation of the cartilages of the fourth ribs with the sternum.

*The left auriculo-ventricular orifice* is on the same level, but on a plane posterior to the right; it lies behind the cartilage of the fourth left rib, near the sternum.

*The pulmonary valves* are on a line with the space between the cartilages of the second and third ribs, to the left of the sternum, and very close to this bone. In some instances they lie a little lower down—viz., on a line with the junction of the cartilage of the third left rib with the sternum, and immediately under it.

*The aortic valves* lie behind the sternum, on a line with the junction of the cartilages of the third ribs with the sternum, and towards the left edge of this bone. When the valves of the pulmonary artery are situated lower down, the semilunar valves of the aorta will be lower also, and on a line with the interval between the insertion of the cartilages of the third and fourth ribs.

The free edge of the semilunar valves of the aorta corresponds accurately, M. Gendrin observes, to the base of the pulmonary valves. A line drawn across the inferior margin of the third ribs corresponds to the base of the valves of the pulmonary artery, and to the free border of the aortic valves.

The right ventricle ascends higher than the left, and the left ventricle descends lower than the right. Hence the origin of the pulmonary artery is on a plane above that of the aorta.

The pulmonary orifice is the highest up, as well as the most anterior, of all the orifices of the heart. The aortic orifice lies behind it, and on a plane lower down. The left auriculo-ventricular orifice is immediately behind the aortic orifice, but on a plane lower down. The right auriculo-ventricular orifice is nearly on the same plane as the left, but more anterior.

*The Aorta.*—The ascending portion of the arch of the aorta

comes to the right of the sternum, between the cartilages of the second and third ribs. In this part of its course it is within the pericardial sac, and in the dead subject lies at the depth of one inch and a half from the surface, the margin of the right lung and the pericardium being between it and the parietes of the chest. The transverse portion of the arch of the aorta crosses the trachea at the centre of the first bone of the sternum, on a line with the lower margin of the articulation of the cartilages of the first ribs with the sternum, and at a still greater depth from the surface. The arch of the aorta approaches most closely to the parietes at the point at which the arteria innominata comes off; that is, on a line with the junction of the cartilage of the second right rib with the sternum.

*The pulmonary Artery.*—The origin of the pulmonary artery is on a line with the junction of the cartilages of the third ribs with the sternum; the tip of the left auricle resting against its left side; it ascends about two inches before it divides; and a portion of the margin of the vessel here comes to the left of the sternum, between the cartilages of the second and third ribs. The division of the artery is on a line with the upper edge of the cartilage of the second ribs, where they join the sternum; the apex of the pericardial sac being on a line with the junction of the cartilages of the second ribs with the sternum, though it is sometimes higher up, and on a line with the cartilage of the first ribs.

## CHAPTER III.

**MOTIONS OF THE HEART.—FREQUENCY OF THE HEART'S MOVEMENTS, AND OF THE PULSE.—EFFECTS OF POSTURE ON THE PULSE.—MECHANISM OF THE ACTION OF THE VALVES.**

THE heart we have seen to be composed, essentially, of muscular tissue. As a muscular organ, it is adapted to perform certain motions. These consist in alternate contractions and relaxations of its muscular fibres; but the heart being a hollow viscus, and divided in its interior into distinct chambers, the contraction and relaxation of its muscular parietes must necessarily occasion the diminution and enlargement of its cavities, and of course react upon the blood they contain. The ventricles are the parts of the heart upon which the circulation mainly depends; the auricles (as was remarked by Hunter) are to be considered rather as reservoirs, “capable of holding a sufficient quantity of blood to supply the ventricles, and ready always to fill them as they are in need of it.”

The function of the heart, it is scarcely necessary to observe, is the circulation of the blood; it is the principal agent in this important process; and, as life depends upon its regular and constant action, the motions of the organ are altogether independent of the will. The heart, therefore, considered as a muscular viscus, belongs to the class of involuntary muscles, its movements being not only uninfluenced by the will of the individual, but carried on without his consciousness.

The manner in which the motions of the heart are performed the order in which they succeed each other, and the phenomena which characterise its movements, have been determined by an examination of the heart in animals, where the parietes of the thorax had been removed and the heart exposed, the animal having been previously deprived of sensation, and artificial res-

piration kept up. The opportunity has likewise been occasionally afforded of examining this organ in the human subject in certain rare cases of congenital malformation, where an infant was born with a deficiency of the anterior parietes of the thorax, and the heart presented itself naked and uncovered to the view. Few parts of our subject have been more frequently or more carefully investigated than this, and there are few upon which a greater amount of time, labour, and talent have been bestowed.

The motions of the heart consist, as I have said, of alternate contractions and relaxations of the parietes of its several cavities. The contraction of the ventricles, or their systole, as it is commonly termed, constitutes their active state; as soon as this ceases, the muscular tissue relaxes, their cavities enlarge, and return to a state of fulness; when the ventricle is said to dilate, or its diastole as it is termed, occurs. By the ventricular systole, the blood is propelled into the aorta and pulmonary artery; as the ventricles re-expand, the blood, favoured by gravity, passes in a full stream from the auricles into the ventricles, to be again expelled when the ventricular systole ensues.

From the Report of the Committees of the British Association for 1838-39 and 1839-40, it appears that "the visible systolic and diastolic motions are first perceived at the bases or fixed parts of the cavities; viz. in the auricles at the sinuses, and in the ventricles at the fundis cordis; and that the apices of the auricles and ventricles, or free parts, are brought into full action after the others, and only just before the supervention of the opposite and next succeeding condition of the cavities respectively, whether that condition be systole or diastole."

It will now be necessary to consider separately the motions of each of the cavities of the heart.

*Auricular Systole.*—The systole of the auricles is a quick, short, sudden motion. Lower says its rapidity equals the explosion of gunpowder. It immediately precedes the ventricular systole, the one motion appearing to be propagated into the other. It commences in the sinus, and extends to the appendix, but by a very minute interval. According to some authorities, it is first observed in the appendices. In feeble states of the heart's action it becomes vermicular.

The auricular systole is most apparent in the appendices, and

is very feeble compared with that of the ventricles. By it a small additional quantity of blood is propelled into the ventricles; but its contraction is too slight either to empty the auricular cavities or to cause the dilatation of the ventricles. In the majority of cases, likewise it is too feeble to produce sound. Nevertheless, in some of the experiments of the Committee of the British Association, and in those conducted in America by Drs. Pennock and Moore, sound, though faint, was sometimes heard, which, in a great degree, merged into that of the ventricular systole, but still was real. The auricular systole occurs towards the end of the period of repose of the heart; its duration is about the eighth part of an entire beat of the heart.

*Auricular Diastole.*—The diastole of the auricles is a passive movement; these cavities are gradually and progressively distended, from the sinus to the appendices, by the blood which enters from the venæ cavæ and pulmonary veins. It continues from the termination of one auricular systole to the commencement of the next, so that it persists through the systole of the ventricles, during their diastole, and during part of the interval of repose. M. Cruveilhier states, that the right auricle during its diastole (in a case of ectopia of the heart, which he had an opportunity of examining,) seemed ready to burst, so distended was it, and so thin its parietes; the left did not exhibit the same appearance, at least so decidedly. The duration of the diastole of the auricles is about seven-eighths of an entire beat of the heart.

*Ventricular Systole.*—The systole of the ventricles is a gradual, progressive, and gliding movement, and performed with great force: it commences suddenly, instantly succeeding the auricular systole, so that the one movement appears to be propagated into the other. During their systole the parietes of the ventricles become pale, hard, and convex; the vertical and transverse diameters are diminished, the apex is approximated to the base, and describes a spiral motion from right to left, and from behind forwards, coming in contact with the parietes of the thorax between the cartilages of the fifth and sixth ribs on the left side, where the impulse of the heart is felt. Dr. Sibson\* (in some experiments made by him) says, the ventricles in contracting, felt “like the pushed, revolving point of a pencil.” “Wherever (he observes)

\* Trans. of Prov. Association, vol. xii.

“the junction of the left ventricle to the auricle could be observed, the apex and base of the ventricle were seen to approach each other steadily during the whole systole; so that at the end of the systole, the apex was about the third of an inch nearer the left auricle than at the beginning, and the aorta was drawn downwards about a quarter of an inch.”

In the experiments performed by Drs. Pennock and Moore,\* the apex of the left ventricle was not observed to be approximated to the base during its systole. The expulsion of the blood from the ventricle (they observe) is effected by an approximation of the sides of the heart only, and not by a contraction of the apex towards the base: during the systole, the heart performs a “spiral movement, and becomes elongated.” All other observers have, however, noticed the shortening from above downwards of the ventricular portion of the heart during the systole; and from the manner in which the fleshy columns of the valves are inserted, such would appear to be essential to the perfect action of the auriculo-ventricular valves, when the ventricles contract.

The phenomena presented by the systole of the ventricles have been well described by M. Cruveilhier,† as observed by him in a case of ectopia of the heart, in an infant which lived for about fourteen hours.

“During their systole the ventricles become pale, their surface wrinkled, the superficial veins swollen, and the spiral fibres which form the apex of the heart become more evident. At the same time the ventricles diminish in all their diameters, the appearance of shortening being most perceptible in the vertical diameter.”

“During the systole of the ventricles, the apex of the heart (that is, of the left ventricle) describes a spiral or turnscrew motion from right to left, and from behind forwards. This spiral movement is slow, gradual and as it were, successive; and it is to it that the impulse of the apex of the heart is due. The ventricular systole is not accompanied by a motion of projection of the heart forwards; it is the spiral contraction exclusively which projects the apex of the organ against the parietes of the thorax.”

The ventricles do not appear to empty themselves completely during their systole; this, however, cannot be ascertained with

\* Medical Examiner, Nov. 1839.

† Gazette Medicale.

certainly, owing to the thickness of their parietes; but, as Dr. Hope observes, the diminution of their volume is not in general so great as to convey the impression that they do. During the ventricular systole the blood is propelled with considerable force into the arterial orifices, the first sound of the heart is heard, and the impulse and pulse are felt. The systole of the ventricles occupies about one-half the period of an entire beat of the heart; that is, it is as long as the diastole and the period of repose together, and is double the length of the ventricular diastole.

*Ventricular Diastole.*—The diastole of the ventricles instantly succeeds the systole; it is a sudden and instantaneous movement; in it the ventricles increase in all their dimensions, the apex recedes from the base, the heart becomes elongated, the surface smooth, and it assumes the shape and position which it had previous to the systole. This movement of the heart has been divided into two stages; “the first, which immediately follows the ventricular systole, is sudden, the apex being pushed downwards, and *apparently* passing deeper into the chest, and is occasioned by the return of the heart to its state of rest.” “The second is also sudden, and attended by a rapid but not very extensive enlargement of the heart in all its dimensions.” The force with which the ventricular diastole is accomplished is very considerable: in large animals, the hand grasping it is forcibly opened.

M. Cruveilhier gives the following account of the phenomena observed by him in the case of the infant before alluded to:

“The ventricular diastole was sudden and instantaneous; the ventricular cavities became filled, swelled out, elongated, and the apex was projected downwards: it seemed at first sight as if this constituted the active movement of the organ, so rapid and energetic was it. One forms no idea of the force with which the dilatation overcomes any pressure upon the heart; the hand closed round it is violently opened.”

“The ventricular diastole is accompanied by a movement of projection of the heart downwards, which was carried to the maximum when the infant was placed vertically. This motion was so decided that at first I was inclined to think that it was during the ventricular diastole that the impulse of the heart against the thoracic parietes occurred.”

During the ventricular diastole the blood passes in a full stream.

from the auricles into the ventricles: it is not impelled by the auricles, neither is it the entrance of the blood which causes the ventricles to dilate; the utmost force of the auricular contraction could not cause the dilatation of the ventricles. Besides, the auricular systole does not occur until after the ventricular diastole. The duration of the diastole of the ventricles is brief compared to that of the systole, being about half the length of the former movement, and about one-fourth of the period of an entire beat of the heart; the second sound of the heart is synchronous with it.

*Order of succession of the Heart's motions.*—The systole precedes the diastole in both the auricles and ventricles. The systole of the right and left auricle is synchronous, as is their diastole. The systole of the two ventricles is also synchronous, as is their diastole.

The systole of the auricles occurs during the latter part of the period of repose of the ventricles. The diastole of the auricles occupies the remainder of the period of repose of the ventricles, and the entire period of the ventricular systole and diastole. The systole and diastole of the ventricles occurs at the same period as the auricular diastole.

The order in which these several motions succeed each other is as follows:

1. The auricular systole.
2. The ventricular systole.
3. The ventricular diastole.
4. The period of repose of the ventricles; during the latter part of which period the auricular systole occurs again.

From the termination of the diastole of the ventricles until the commencement of the next systole, “the ventricles are in a state of perfect repose, their cavities remaining full but not distended.”

If the duration of these movements be divided into four equal periods, the ventricular systole would, according to Laennec, occupy about two; the ventricular diastole something more than one; and the interval of repose something less than one. Or in portions of a second, the ventricular systole would occupy about half a second; the diastole a little more than a quarter of a second; and the period of repose something less than the quarter of a second.



Dr. Williams divides the period, from the commencement of one pulse to the commencement of another, into five equal parts. Of these the two first are occupied by the ventricular systole, one-fifth is occupied by the diastole, in part of which occurs the short second sound. The period of natural rest and silence succeeds, occupying the remaining two-fifths.

If the duration of the movements of the auricles be divided into eight equal parts, the auricular systole would occupy about one-eighth—their diastole the remaining seven-eighths : the auricular systole occupying part of the period of the repose of the ventricle ; the auricular diastole occupying the remainder of the period of repose, as well as the period of the ventricular systole and diastole.

*Periods of repose of the Heart.*—When the heart's action is very rapid, the alternate movements succeed each other so quickly that there appears to be no interval of repose. On the other hand, when it is very slow, two distinct intervals are distinguished—one short between the systole and the diastole of the ventricles ; the other longer, between the diastole and the next succeeding systole. The first of these intervals M. Gendrin proposes to call the *peri-systole* ; the second, the *peri-diastole*. According to MM. Barth and Roger, the shorter period of repose has the same duration as the diastole of the ventricles ; and the longer period of repose, about the same duration as the ventricular systole ; and, if the whole period be divided into six equal parts, the systole would occupy about two, the short period of repose one, the diastole one, and the long period of repose about two. According to Dr. Walshe,\* when the heart is beating with moderate frequency, as 60 in the minute, if the period of an entire revolution of the organ be divided into ten equal parts, the systole would occupy four, the post-systolic silence one, the diastole two, and the post-diastolic silence three. It would therefore appear that the period of repose of the ventricles, and of their active state, has about the same duration, which would give about twelve hours' rest out of the twenty-four to the muscular fibres of the ventricles. But, as the muscular fibres of the ventricles are relaxed during their diastole, the period occupied by it may be added, which will make the period of repose or rest of the ventricles considerably longer. As the auricular systole

\* On Diseases of the Lungs and Heart.

occupies only one-eighth part of an entire beat of the heart, and the diastole the remainder, the auricles (if the period of their diastole be considered as a state of repose,) may be said to be at rest for twenty-one out of the twenty-four hours.

#### FREQUENCY OF THE HEART'S ACTION, AND OF THE PULSE.

The rapidity of the heart's action is not the same at every period of life : it varies also according to the sex, the temperament, and the idiosyncrasy of the individual; it differs at different periods of the day, and in different positions of the body, and it is remarkably influenced by affections of the mind.

In the female, the heart's action is more rapid than in the male; the pulse of the adult female exceeding in frequency the pulse of the adult male, of the same mean age, by from nine to ten beats. According to Dr. Guy,\* "the average pulse of the adult male may be stated at 70; that of the adult female at 80." "The female pulse (he observes) differs little from the male pulse during the first seven years of life; but after seven years of age the mean pulse of the female exceeds that of the male by from six to fourteen beats, the average excess being nine beats."

In early life, the heart's action is much more rapid than in the adult; and in the adult it is somewhat more rapid than in advanced life. Thus, while in infancy the number of the pulsations of the heart averages from 120 to 140 in a minute, the number in old age is usually between 50 and 70. The following table, given by Müller,† shows the average frequency of the heart at different ages :

In the embryo, the number of beats in a minute is	.	.	.	150
Just after birth	.	.	.	from 130 to 140
During the first year	.	.	.	" 115 „ 130
„ „ second year	.	.	.	" 100 „ 115
„ „ third year	.	.	.	" 90 „ 100
About the seventh year	.	.	.	" 85 „ 90
„ „ fourteenth year	.	.	.	" 80 „ 85
In the middle period of life	.	.	.	" 70 „ 75
In old age.	.	.	.	" 50 „ 65

It was formerly supposed that the rapidity of the heart's action gradually and progressively diminished from birth upwards; that it was quickest soon after birth; and that there was a gradual and

\* Cyclopædia of Anatomy and Physiology. † Elements of Physiology, vol. i.

sensible diminution in its frequency after that period. The remarkable fact has, however, been ascertained, that the pulse of the infant soon after birth is not unfrequently as slow as that of the adult. M. Billard\* first called attention to this phenomenon, which has also been investigated and corroborated by M. Valleix.† According to him, the mean frequency of the pulse in thirteen healthy infants, from two to twenty-one days old, when asleep, averaged 87 in a minute; when awake, from 90 to 100: but it was readily excited by motion or emotion. From the seventh to the twenty-seventh month, the average was 119 for the male, and 124 for the female infant; and it continued a little above 100 up to the age of six years.

According to Mr. Gorham,‡ the mean frequency of the pulse in sixteen infants under one day old was 123; in forty-two observations on infants from one to seven days old, the average was 128. Dr. Guy says, the observations of MM. Billard and Valleix are by no means borne out by his experience. "There is reason to believe (he thinks) that these low frequencies of the pulse of infants occur in that state and degree of debility, without disease, which gives rise to an infrequent pulse in the adult, and that they do not occur in strong and vigorous health."

The heart's action is less rapid during sleep than when awake; this is probably connected with posture. The decrease in frequency of the heart's action occasionally observed in the foetus in utero, has been attributed to the sleep of the embryo. The action of the heart is much slower when the individual is at rest than during or after bodily exertion. Dr. Bryan Robinson§ states that the pulse of the adult, which ranged when at rest from 64 to 74, rose to 100 when walking at the rate of two miles an hour; to 140 when walking at the rate of four miles an hour; and when running fast, to 150 or more.

The heart's action is usually slightly accelerated after meals, particularly after the use of warm or spirituous liquors; and contrary to the opinion at one time entertained, it is perceptibly quicker in the morning than in the evening. Dr. Knox,|| who was the first to establish this fact, says the pulse is quicker by

\* *Traité des Maladies des Enfants.* † *Mem. de la Soc. Med. d'Observation*, T. 2.

‡ *Medical Gazette*, vol. xxi.

§ *Treatise on the Animal Economy*, 3rd edition.

| *Ed. Med. and Surg. Jour.* vol. xi.

eleven beats, on an average, in the morning than in the evening. Dr. Guy's \* experiments corroborate those of Dr. Knox. In the article "Pulse," in the Cyclopædia of Anatomy and Physiology, he has given a series of tables showing the diurnal variations of the pulse in the two sexes; "the facts contained in which may (he observes) be taken to establish the general law first set forth by Dr. Knox, that the pulse is less frequent in the evening than the morning; but it is obviously subject to numerous exceptions."

*Effects of posture on the Pulse.*—Posture has a remarkable influence upon the rapidity of the heart's action: it is more frequent in the sitting than in the recumbent posture; and more frequent in the erect than in the sitting posture. Attention was first called to this remarkable fact above a hundred years ago, by Dr. Bryan Robinson, of Dublin, in his "Treatise on the Animal Economy." In the third edition of this work, published in the year 1738, he states that the pulse, which in the recumbent posture was sixty-four, rose to sixty-eight in the sitting posture, and to seventy-eight in the erect posture. The late Dr. Macdonnell, of Belfast, so long ago as the year 1784, determined experimentally this influence of change of posture on the pulse; he termed it "*the differential pulse*:" the results of his observation were communicated to the profession at the meeting of the British Association in Dublin. This phenomenon has also been investigated with much care by Dr. Graves of Dublin, by Dr. Knox of Edinburgh, and by Dr. Guy of London.

According to Dr. Graves,† the difference between the pulse in the erect and recumbent posture in the healthy adult is from six to fifteen beats in a minute: "if the pulse is but sixty, the difference is generally not more than six or eight, and this difference increases with the frequency of the pulse at the time of the experiment: thus, if it has been raised to 90 or 100 by moderate exercise, it is not unusual to find the difference twenty or thirty." That it is not the muscular exertion necessary to raise the body from the recumbent to the erect posture which occasions this difference, was proved, Dr. Graves says, by altering the position without the necessity of any muscular exertion on the part of the person experimented on. He tried also the experiment of inverting the position, placing the person with the head downwards and t/

\* Guy's Hospital Reports, No. ix. † Clinical Medicine, 2nd edition.

feet in the air: the frequency of the pulse was not, however, altered, but its strength diminished often very considerably, and it not unusually became irregular.

Dr. Knox\* has shown that the increase in frequency of the pulse occasioned by change of posture, is different at the different periods of the day. "During the morning, the mere change of posture from the horizontal to the erect will increase the pulse by about fifteen or twenty beats; at mid-day, this increase will be ten; and in the evening, only four or six."

Dr. Guy instituted an elaborate series of experiments to determine the alterations effected on the pulse by change of posture. The following are some of his conclusions† under this head:

"1. In the healthy adult male, the mean Nos. of the pulse are—standing, 79; sitting, 70; lying, 67.

"2. In the healthy adult female, the Nos. are—standing, 89; sitting, 82; lying, 80.

"3. In both sexes, the extremes are very remote from the mean results, and the exceptions to general rules very numerous.

"4. In both sexes, also, the effect of change of posture increases as the frequency of the pulse increases.

"5. The effect of change of posture on any given frequency of the pulse, is much greater in the male than in the female.

"6. The effect of change of posture on the pulse is greater in the forenoon than in the after part of the day.

"7. The inverted posture of the body lessens the frequency of the pulse.

"8. The varying frequency of the pulse in different postures of the body is due to muscular contraction."

In states of debility, as in patients recovering from fever, the difference between the frequency of the pulse in the erect and recumbent postures becomes (Dr. Graves observes) a useful guide in the treatment: "the greater the difference the greater the debility." In hypertrophy, with dilatation of the ventricles, on the other hand, Dr. Graves found the pulse to be very little altered with respect to frequency on changing the position. In anæmic and chlorotic females, and in individuals who have suffered from profuse hæmorrhage, I have observed that the difference between

\* Edinburgh Medical and Surgical Journal, vol. xi.

† Cyclopædia of Anatomy and Physiology—article, Pulse.

the frequency of the pulse in the recumbent and sitting postures is very marked; but these morbid states are always accompanied by more or less debility; and the pulse, in respect to frequency, is always above the normal standard.

*Cause of the Influence of Change of Posture on the Pulse.—*

With respect to the cause of this alteration in the frequency of the pulse in different positions of the body, writers are not agreed. Dr. Guy refers it exclusively to muscular contraction; by others it has been attributed to the altered position of the heart, its valves and orifices in the different positions; while Dr. Graves says “he cannot advance even a plausible conjecture concerning the reason why a change of posture should so affect the frequency of the pulse.” “The two postures (Dr. Guy observes) between which there is the most marked difference in the frequency of the pulse, viz. the erect and sitting postures, are precisely those in which there is no difference in the position of the heart or its valves, and very little difference in the resistance offered to the circulation; while the sitting and recumbent postures, between which there is so slight a difference in the number of the pulse, are accompanied by a marked change in the position of the heart and its valves, and of the column of blood to be propelled. On the other hand, the difference in the amount of muscular contraction, required to support the body, in the erect and sitting postures is much more considerable than that required to support the body in the sitting and recumbent positions—differences in strict conformity with the observed frequencies of the pulse in the several postures.”

Dr. Wardrop\* accounts for the difference of the pulse in the erect and recumbent postures by his ingenious theory of the *musculo-cardiac function*. According to him the contraction of muscles not only accelerates the flow of blood in the veins towards the heart, but the compression exercised on the arteries by the same contraction of the muscles impedes more or less the flow of blood in the arteries; the cavities of the heart thus receive a larger amount of blood, and as the energy of the heart's action depends within certain limits, upon the quantity of blood it contains, it will be accelerated by the change from the recumbent position, in which the muscles are relaxed, to the sitting or erect posture, in which many of the muscles are in a state of contraction.

\* Treatise on Diseases of the Heart.

It appears to me, that writers, in seeking to explain the influence of position upon the pulse, have erred in endeavouring to refer it exclusively to a single cause: it probably lies more in the lungs than in the heart; the latter organ being indirectly excited in consequence of the increased frequency of the respiration, while it is very probably favoured by muscular contraction. For instance, the chest expands less, and less air is taken in in inspiration, in the recumbent than in the erect or sitting postures; there is less of the feeling, likewise, which inclines to frequent renewal of the air in the former than in the latter position. In certain diseased states, this becomes sufficiently obvious: whenever dyspnœa is urgent, no matter what may be its cause, the patient is unable to remain in the recumbent posture, and seeks to obtain ease by sitting up. As there is less necessity for the blood to circulate quickly through the lungs in the recumbent than in the erect or sitting posture, the respirations are less frequent, and the general circulation is likewise less rapid. At the same time, there can be no doubt that the venous blood is returned, from the head and upper extremities, to the right side of the heart more quickly and more freely in the erect and sitting than in the recumbent posture, being remarkably favoured by gravity in the former positions; the pulmonary circulation and the respirations necessarily, therefore, increase in frequency; the left side of the heart then feels its influence, the blood being conveyed more rapidly to it, and the general circulation becomes quickened in the same ratio. Hence there is no difficulty in understanding why the pulse should be more rapid in the one position than the other. Its increased rapidity in the erect over the sitting posture is no doubt favoured by muscular contraction.

*Ratio of the Pulsations of the Heart to the Respirations.*—Under ordinary circumstances, the proportion which the respirations bear to the pulsations of the heart is about as *one* to *four*; this was the ratio established by MM. Prevost and Dumas: the average number of respirations in a minute, in a healthy adult, being between sixteen and twenty, and the number of pulsations of the heart, between sixty-four and eighty. When the heart's action is accelerated by motion or by exercise, the respirations are accelerated in the same ratio; and when its action becomes slow again, the respirations become also less frequent.



Mr. Hutchinson\* has given a table of the number of respirations, in a minute, in 1714 healthy adult males, when in the sitting posture; from which it appears that the majority make between sixteen and twenty-four respirations in a minute—of these, a large number make twenty; and in the greater number there were four beats of the heart for one inspiration. The following table, given by Dr. Guy, shows the average proportion of the pulse to the respiration in 238 experiments; the pulse varying from 44 to 85 beats, and the respirations from  $15\frac{1}{2}$  to  $20\frac{1}{2}$ : the majority of the experiments were made in the sitting posture:

No of Observations.	Pulse.	Proportion.
8 . . . . .	45-50 . . . . .	2.75 to 1
37 . . . . .	50-55 . . . . .	3.05 to 1
50 . . . . .	55-60 . . . . .	3.31 to 1
50 . . . . .	60-65 . . . . .	3.52 to 1
50 . . . . .	65-70 . . . . .	3.59 to 1
27 . . . . .	70-75 . . . . .	3.82 to 1
12 . . . . .	75-80 . . . . .	4.18 to 1
4 . . . . .	80-85 . . . . .	4.31 to 1

The following table† gives the average result of some experiments made on himself, by Dr. Harden, in order to determine the ratio of the pulse to the respiration in different postures:

	Standing.	Sitting.	Lying.
Pulse . . . . .	80 . . . . .	70 . . . . .	66
Respirations . . . . .	16 . . . . .	14 . . . . .	12

According to M. Parrot,‡ the frequency of the heart's action increases in a corresponding ratio with the elevation above the level of the sea. Thus—

When the pulse at the level of the sea was 70			
at 3282 feet above its level it rose to	75		
at 4875	82		
at 6500	90		
at 8125	95		
at 9740	100		
at 13000	110		

The cause of the heart's action increasing in frequency according to the elevation above the level of the sea, appears to lie in the lungs rather than in the heart. We know that as the air becomes more rarified the respirations become more frequent; the heart, therefore, is only indirectly excited in consequence of the increased

\* Med. Chir. Trans., vol. xxix.

† Amer. Jour. of Med. Sciences, vol. v., 1843.

‡ Müller's Physiology, vol. i.



frequency of the respiration, and the ratio between the number of respirations and the number of pulsations of the heart is, probably, the same at each level.

Staff Surgeon Hunter,\* whose opportunities for observation have been very extensive, states, on the other hand, that "he has never noticed the least difference in the pulse from elevation, whether at Madras, or Bombay, on the level of the sea; Poona and Belgaum, 2000 feet above it; Candahar, 4000; Kelat or Cabul, 6000; or Ghuznee, or along the range of the Suliman mountains, 8000, or 9000." "Along the latter," he says, "we several times changed our elevation, from 1000 to 2000 feet in the day; and I cannot but think," he adds, "that the presumed difference has been originally based upon theory, and continued without further inquiry."

Several pathological conditions have great influence upon the frequency of the pulsations of the heart: in but a few it becomes slower; in the large majority of diseases it becomes more rapid than natural; while in certain morbid states the ratio between the number of the respirations and the number of pulsations of the heart becomes altered. These are matters, however, for subsequent consideration.

*Rapidity of the Passage of the Blood through the System.*—The rapidity with which the blood passes through the system can be estimated, if the amount which is transmitted into the aorta at each systole of the left ventricle is known, and if the entire quantity of blood contained in the vessels is determined—the number of pulsations of the heart being given. Thus it has been estimated that the whole amount of blood contained in the vessels is about twenty-eight pounds; and that an ounce and a-half is expelled at each systole of the left ventricle. If, then, the heart of the adult beats seventy-five times in a minute,  $112\frac{1}{2}$  ounces, or a little more than seven pounds of blood, would pass through the ventricle in a minute; in four minutes the entire twenty-eight pounds would pass through the heart; and in every hour it would pass through it fifteen times.

*Force with which the Blood is Propelled by the Left Ventricle.*—The force with which the blood is expelled by the left ventricle has been variously stated by physiologists. Hales made many

\* London Medical Gazette, August, 1850.

experiments, with the object of determining this point; he estimated the force with which the blood is propelled into the aorta at four pounds six ounces: "seven feet and a half being the height to which he supposed the blood would rise in a tube connected with the large arteries." M. Poiseuille invented an instrument for measuring the momentum of the current of blood in the large arteries, to which he gave the name of *hemadynamometer*, from which he estimated the force with which the blood is propelled into the aorta at four pounds three ounces; "a result which agrees remarkably with that obtained by Hales."

In employing compression for the cure of external aneurism, I have found that the weight which was sufficient to diminish materially, or to check the current in a large artery, differed considerably in different subjects: in some instances a pressure equal to four pounds upon the femoral artery in the groin was sufficient to check the pulsation of a popliteal or femoral aneurism; in the majority of cases, a pressure equal to six or eight pounds was necessary to effect the same object; while sometimes a weight of ten pounds was requisite.

#### MECHANISM OF THE ACTION OF THE VALVES.

The valves at the arterial orifices of the heart are, Hunter\* observes, "inelastic membranes having no action within themselves;" they are raised or depressed simply by a mechanical process, which can be effected equally well in the dead as in the living heart. The valves at the auriculo-ventricular orifices, on the other hand, being connected to the parietes of the ventricles by muscular columns, the shortening or lengthening of which are essential to the due performance of their valvular functions, cannot be made to act perfectly in the dead subject.

*Auriculo-ventricular Valves.*—It has been already stated that the auriculo-ventricular valves are composed of curtains, of tendinous cords connected with these curtains, and of fleshy columns which are attached by one extremity to the tendinous cords, and by the other to the parietes of the ventricle. Anatomists, in describing the mode of action of these valves, state, that one use of the fleshy columns is, by their contraction, to draw down the curtains of the valve, and so to approximate the edges of the valve

\* Treatise on the Blood.

and close the orifice. It must, however, be recollected that, at the period at which the auriculo-ventricular valves close their orifices, viz., at the very commencement of the ventricular systole, the ventricles are full of blood; hence the contraction of the carneæ columnæ, and the tension of the tendinous cords, instead of approximating the edges of the valves and closing the orifices, would tend rather to hold the curtains of the valves open, because the fleshy columns all run, more or less, towards the apex of the heart to be inserted into the parietes of the ventricles.

It was long since remarked by Senac, that “in order for the auriculo-ventricular valves to close their respective orifices, the apex of the ventricle must be approximated to the base, as in the dilated state of the ventricle they are too short to do so.” Hunter in describing these valves, observes—“the tendinous cords are inserted into muscular columns, the intention of which is very evident; for if they had gone the whole length in the form of tendon, they would have been too long when the ventricle contracted, and the valves in such a case would have allowed of being pushed into the auricles; but the carneæ columnæ keep the valves within the ventricle in its contracted state.” The use of the carneæ columnæ appears, then, simply to be to adapt the valve to the altered state of the ventricle during its systole and diastole, and to prevent them from being reversed during the systole. When the apex of the ventricle is approximated to the base during the ventricular systole, if these valves were not provided with muscular columns which shorten as the ventricle shortens, the valves would be reversed; but from the direction of the fleshy columns, as the ventricle is shortened, the carneæ columnæ must be shortened at the same time.

The use of the carneæ columnæ therefore evidently is, not to close the curtains of the valves; this is effected by the mechanical operation of the blood contained in the ventricle, as was long since shown by Lower, though recently advanced as a novel view by Baumgarten and Hamernjk. The blood which enters from the auricle during the ventricular diastole, falls by its own gravity to the bottom of the ventricle, the curtains of the valve are thus floated towards the base of the ventricle; the auricular systole then impels a small additional quantity of blood into the ventricle, the systole of the latter instantly ensues, and the curtains of the

valve are applied and pressed together so as to close the orifice. Hence the blood which enters the ventricle becomes, in a great measure, the means by which the auriculo-ventricular valves are closed, and regurgitation prevented.

The curtains of the mitral valve in closing the orifice, “meet (Mr. T. W. King \* observes) not only by their free edges, but by the marginal portions of their auricular surface; and thus on each curtain there is a broad line or surface of contact situated just within the auricle, or between it and the ventricle, varying in breadth, regularity, and distinctness.” These he terms “the surfaces of contact or attrition;” very slight traces of them are discoverable in the healthy adult heart.

As soon as the ventricular systole ceases, the muscular fibres forming the walls of the ventricles, and the *carneæ columnæ* of the auriculo-ventricular valves, are relaxed; the curtains of these valves, no longer pressed upwards by the blood, separate from one another: this fluid enters in a full stream from the auricle until its systole again ensues, when the phenomena mentioned before are repeated.

*Sigmoid and Semilunar Valves.*—The mechanism by which the valves at the orifices of the aorta and pulmonary artery close their respective orifices, is, as I have said, simply mechanical; behind each of these valves a little sinus (*sinus of Morgagni*) is seated, into which the blood readily insinuates itself as the ventricular systole ceases; and the valves are pressed down by the weight of the column of blood above. Without some provision of this kind, the blood could with difficulty get behind those valves, and regurgitation into the ventricles, of necessity, would frequently take place during the diastole of these cavities.

When the ventricular systole ensues, the sigmoid and semilunar valves are elevated and pressed against the sides of the aorta and pulmonary artery, by the blood in its passage into these vessels; and here again another use of the sinuses of Morgagni comes into operation—viz., to afford a space for the valves to lie back in, at this period of the heart’s action, and thus to render the interior of the aorta and pulmonary artery perfectly smooth and even at the part. If there had not been some provision of this kind, these valves would project, in a certain degree, upon the interior of the

\* Guy’s Hospital Reports.

artery, render this portion of the vessel uneven, and of course increase the friction between the blood and the interior of the artery in its passage through it. When the aortic valves become rigid from disease, they cannot lie back in their normal position during the ventricular systole, and not only is the blood in its passage from the ventricle into the aorta impeded, but regurgitation into the ventricle is, as we know, frequently permitted.

The little fibro-cartilaginous bodies (*corpora Arantii*) which are situate near the centre of the free margin of each of the semi-lunar valves, are supposed by some anatomists to serve the purpose of filling up the small space, which they suppose would be left in the centre, when the valves fall down; but, as these little bodies are not seated at the very margin of the valves, they could scarcely effect this object, which indeed is unnecessary, because it has been shown by Dr. Duncan that the arterial valves, particularly those of the aorta, overlap one another, indeed, Mr. King says "the surfaces of contact are not unfrequently equal in extent to the remainder of the curtain of the valve." The use of these little bodies evidently is to strengthen the valves, by serving as points of insertion for the tendinous bands which enter into their formation. Without this provision these valves would be very liable to become reversed.

*Safety-valve Function of the Tricuspid Valve.*—Mr. Hunter,\* in describing the action of the tricuspid valve, observes—"I have reason to believe that the valves on the right side of the heart do not so perfectly do their duty as those of the left; therefore we may suppose it was not so necessary." Mr. Adams,† after quoting the foregoing remark of Hunter, observes—"This circumstance, in my opinion, has not been sufficiently noticed, nor the influence that such a structure may have on the circulation in its natural or morbid state, considered. I look upon this difference in the valves of the right and left side of the heart to be a natural provision to allow of a partial reflux into the right auricle on those occasions when, from any cause, the passage of the blood through the arterial opening is retarded. Such a provision was absolutely necessary in the right or pulmonary ventricle, as various natural causes must momentarily retard the passage of the blood through the lungs."

\* *Treatise on the Blood.*

† *Dublin Hospital Reports*, vol. iv.

More recently, Mr. King,\* in an elaborate paper on "the Safety-valve Function in the Right Ventricle." has entered at length upon the subject, and pointed out the mechanism in the construction of this valve by which he considers regurgitation to be permitted. In the same communication he has given a minute description of the tricuspid valve, and of the manner in which its fleshy columns are connected with the ventricular walls. The fleshy columns of the anterior and right division of this valve are principally inserted (he observes) into the yielding wall of the ventricle; those of the posterior division into the septum: the latter is consequently more fixed. The anterior and right divisions of the valve he calls "the curtains of distension," and he considers them the agents by which the orifice is opened so as to permit of regurgitation into the auricle.

In the normal state of the tricuspid valve, and of the orifice of communication between the right auricle and ventricle, and when the ventricle is only moderately distended, regurgitation is not permitted: it does, however, occur if the right ventricle at the commencement of its systole is much distended: this may occur in a state of health when the heart's action is increased by any cause; but it subsides when this ceases.

Dr. Blakiston† objects to the theory that the tricuspid acts as a safety-valve; he thinks the chances of life would not be improved "did nature thus attempt to relieve arterial congestion by means calculated to induce venous engorgement." "When regurgitation takes place to any extent, it is impossible," he says, "to conceive a more powerful obstruction than is thus offered to the passage of the blood from the veins into the heart by a strong counter-current forced backwards by the systole of the right ventricle."

Whatever difference of opinion may exist respecting the mode of action of the valve at the right auriculo-ventricular orifice, there is none as to that on the left side; the mitral valve always perfectly performs its valvular function, and never, unless it has undergone some morbid change, permits regurgitation. Indeed, the effect of disease upon these two orifices is generally quite the reverse: in the majority of cases in which the mitral valve or orifice is altered from its normal condition, the opening is con-

\* Guy's Hospital Reports, vol. ii.

† On Diseases of the Chest.

tracted; while the tricuspid orifice, on the other hand, is more frequently dilated; and the phenomenon known as “jugular pulsation” almost always attends the latter state.

“The mitral valve (Mr. Adams\* observes) so perfectly closes the aperture of communication between the left auricle and ventricle, that in the natural state no reflux whatever is admitted: this, so useful at the right side of the heart, would have been not only useless, but injurious, at the left side of the organ, as we find the general arterial system at all times equally ready to receive the blood during the systole of the left ventricle; and if the mitral valve did not close the left auriculo-ventricular aperture, a great deal of the force of the aortic ventricle would be wasted, whereby it would be incapable of moving the mass of blood which was destined to fill the arterial system.” “Pathologists, in looking (he adds) to the different nature of the lining membrane at the two sides of the heart, as a means of explaining the greater liability of the left side to disease, have perhaps too much overlooked this circumstance—that while, from the unyielding nature of the mitral valve, all reflux into the auricle is prevented, from this very cause, which renders it effective in the circulation, is it exposed to more frequent injury, from which organic disease may arise, and the ventricle to which it belongs becomes more liable to be ruptured by its own efforts.”

\* Dublin Hospital Reports, vol. iv.



## CHAPTER IV.

IMPULSE OF THE HEART.—SOUNDS OF THE HEART.—MECHANISM BY WHICH  
THE NORMAL SOUNDS OF THE HEART ARE PRODUCED.

THE impulse of the heart accompanies the systole of the ventricles, and the first sound of the organ, and has its cause in the apex of the heart coming in contact with the parietes of the thorax. In the healthy subject, with a well formed chest, the impulse is so slight as not to be perceptible to the individual himself; and is visible only in a circumscribed space, between the cartilages of the fifth and sixth left ribs, about mid-way between the left edge of the sternum, and the nipple, and from an inch to an inch and a-half below the latter. It varies, even in the healthy subject, according to several circumstances, as the age and sex of the subject, the size of the chest, the stature of the individual, the amount of subcutaneous fat, the posture, whether recumbent or erect, the state of inspiration or expiration, the amount of distension of the abdomen, and, whether the examination is made after exercise or mental excitement. The mechanism by which the impulse is produced was long a disputed point, and various have been the theories advanced by physiologists to explain it: even the period of the heart's action at which it occurs has been the subject of difference of opinion.

Thus it was at one time maintained, and the theory has been recently revived by Mr. Robert Cartwright\* and Mr. Brown of Cobham, that the impulse occurs during the diastole of the ventricles. This opinion appeared to derive confirmation from the fact that when the heart of a frog is exposed (which will continue to pulsate for a considerable time after being laid bare), the ventricle, during its diastole, is seen to expand, and to approach the parietes; while during the systole the apex is simply approximated to the base. In this animal, therefore, the heart approaches the

\* Lancet, December, 1851, and January, 1852.



parietes during the diastole, not during the systole of the ventricle, and any impulse which is given must be at the period of the ventricular diastole. An experiment was performed by Oesterreicher,\* which consisted in removing the heart of the frog from the body, and laying upon it a substance sufficiently heavy to press it flat, and yet so small as not to conceal the heart from view. He states that during the systole of the ventricle the weight was raised, but that during its diastole the heart remained flat. This experiment has been quoted by Müller and others, to prove that the diastole of the ventricles is not a muscular act, in ignorance apparently of the foregoing peculiarity in the action of the heart in this animal. In warm-blooded animals, however, experiments and observations repeated over and over again have proved that the impulse occurs at the period of the ventricular systole, and that it is due to the apex of the heart coming in contact with the parietes of the chest.

*Mechanism by which the Impulse of the Heart is Produced.—*

It will not be necessary to delay to notice the various theories which have been advanced in order to explain the mechanism by which the impulse of the heart is produced, many of which are founded on erroneous views. It will be sufficient to observe, that during the ventricular systole the walls of the ventricle become more convex upon the surface, the apex of the heart describes a spiral motion from behind forwards, and from right to left: in performing this spiral movement, the apex glides obliquely upon the pericardium, is approximated to the base, comes in contact with the parietes of the thorax in the intercostal space between the cartilages of the fifth and sixth ribs, and thus causes the impulse. Indeed, this part of the heart is naturally so close to the parietes of the chest that no tilting forward of its apex is necessary to produce the slight shock felt at this period.

It was the received opinion until within a few years, that during the diastole of the ventricles the heart receded from the parietes of the chest, and that the impulse was produced by a blow, or shock given to the ribs by its apex, during the systole. Harvey, Haller, Senac, and Hunter, may be quoted as authorities for this theory. The experiments which have been performed upon animals of late years, and the examination of the action of the organ in cases of ectopia of the heart, have, however, shown that

\* Müller's Physiology, vol. i.

this theory has no foundation, and that the heart “does not suffer any changes in consequence of its own efforts (exclusive of the movements of the lungs and diaphragm) except in its shape and size, in the thickness and tension of its parietes, and in the capacity of its cavities,” which are quite sufficient to produce the slight shock felt when the hand is laid on the parietes of the chest.

M. Ritter has recently advanced this as a novel doctrine, in ignorance probably of the results of the experiments of the “Committees of the British Association.” His experiments are entirely corroborative of those previously made. “The portion of the heart’s surface (he observes) which is in immediate relation to the walls of the chest is at all times in close contact with them; and it is impossible that in any of its motions it can act so as to withdraw itself from the thoracic walls, or so as to leave a space between them.” “Being thus fixed, therefore, it follows that, when the heart contracts and assumes a more globular form, it will exert its distending force on the yielding intercostal spaces against which it rests, and will thrust them forwards, so as to produce the impulse. This distending force cannot be exerted with any effect against the unyielding ribs or their cartilages; and, consequently, the impulse is not perceived by the finger placed over the cartilages of the fourth, fifth or any other rib. If the impulse was caused by an actual stroke or blow of the heart against the walls of the chest, it would be perceived on these parts and on the sternum as clearly as it is in the intercostal spaces, and every person would feel the impulse of his own heart just as a pregnant woman feels any violent movements made by the fœtus in utero.”

*Sound sometimes Produced by the Impulse of the Healthy Heart.*—Although in the healthy subject, when the circulation is tranquil, and the heart’s action is normal, no sound is produced by the impulse, yet it occasionally happens that when the heart is excited to increased action—in other words, when palpitation ensues, whether the cause be mental emotion or corporeal exertion, but particularly the former—the apex of the heart does come in contact with the ribs, the patient feels the blow or shock produced by the impulse of his own heart; and this is accompanied by sound, which of course will be heard at the period of the first sound of the heart. In diseased states it is sometimes so loud as to be audible without the stethoscope, and it may be heard at a short

distance from the patient. This point will be again alluded to when we come to consider the abnormal conditions of the heart.

*Site of the Impulse of the Heart.*—The point at which the impulse of the heart is felt in the healthy male is the intercostal space between the cartilages of the fifth and sixth ribs upon the left side, to the sternal side of the nipple, and about two inches below this point. In the female, owing to the habitual use of stays, the impulse is often a little higher up—viz., between the cartilages of the fourth and fifth left ribs; and in the latter months of pregnancy, for an obvious reason, it is felt higher up than natural, and the apex is pushed more to the left side.

The impulse of the healthy heart is naturally slight: it is more marked in the erect than in the recumbent position, because in the latter position the heart, by its own weight, recedes slightly from the parietes of the chest. For the same reason the impulse becomes more distinct if a person leans forward, and more indistinct if he lies upon his right side. In the erect posture the impulse is said to be slightly lower than in the recumbent posture; but the difference, if any, is very trifling. In very fat persons the impulse is scarcely perceptible to the eye or hand; in very lean persons it is very obvious to both. When the lungs are largely developed they will overlap the heart more than usual; when the lungs are small, less of the heart will be covered by these organs: in the latter case, therefore, the impulse will be better marked than in the former.

*Alteration of the impulse in inspiration and expiration.*—In inspiration, particularly on a full inspiration, the impulse of the apex of the heart will be felt lower down than natural, as low as between the cartilages of the sixth and seventh ribs, or, in the epigastrium, between the line of the xyphoid cartilage. This is partly owing to the connection of the heart with the lungs, and partly to the connection of the pericardium with the diaphragm. On a full inspiration the lungs expand from above downwards, as well as from before backwards; and, according to Dr. Sibson, from the manner in which the pulmonary veins are joined to the left auricle, the heart is drawn down by the descent of the lungs. The principal cause of this descent of the heart in inspiration appears to lie rather in the intimate connection of the pericardium with the central tendon of the diaphragm; as the latter descends it

must bring with it the heart; and from the connection of the inferior vena cava with the diaphragm, it must follow the movements of the latter, and draw down the right auricle. On a full inspiration, the impulse, in addition to being lower down, will be less marked than natural, because the lungs, when fully inflated, meet so as almost to cover the heart, and prevent its apex from coming in contact with the parietes of the thorax.

On a forced *expiration*, on the other hand, owing to the ascent of the diaphragm, the impulse of the heart is felt higher up, viz. on a line with the space between the cartilages of the fourth and fifth ribs on the left side; it is likewise more marked than natural, because the heart is less covered by lung. The point at which the impulse of the heart is felt is altered in some diseases of this organ, or of the lungs, or pleura, as well as in diseases of the abdominal viscera. These matters will, however be considered when we come to describe the diseased states of the heart.

*Double Impulse of the Healthy Heart.*—The impulse of the healthy heart has been almost always described as single; Magerie,<sup>\*</sup> however, who attributes the first sound of the heart to the shock of the apex during the ventricular systole, lays it down that the second sound is due to the shock given by the ventricle to the parietes of the thorax during their diastole. “The ventricles in dilating (he observes) in a great measure under the influence of the rapid influx of the blood, give a shock to the anterior parietes on the right side of the thorax, and thus produces the second clear sound.” Dr. Sibson, † in his valuable essay upon the “Changes in the Situation of the Internal Organs,” observes:—“A second impulse is often felt in persons whose lungs are diminished, and whose great vessels come close to the sternum. This is synchronous with the second sound, and must, I conceive, be due to the sudden springing forwards of the walls of the right ventricle after the systole.” In the year 1848, in a communication ‡ upon the subject of “Aneurism of the Aorta,” made to the Surgical Society of Ireland, I called attention to the fact that the impulse of the healthy heart, when the organ is acting vigorously, is double, not single.

The impulse of the healthy heart, I observed upon that occa-

<sup>\*</sup> Elements of Physiology.

† Trans. of Prov. Association, vol. xii.

‡ Dublin Medical Press, vol. xix.

sion, has been always described as single, just as that of aneurism of the arch of the aorta was supposed to be. If we carefully examine this organ, however, when it is beating vigorously, we shall find that a second but slighter impulse is perceptible, which quickly succeeds the other; and, on applying the stethoscope, we shall find that this second impulse accompanies the second sound of the heart; it appears as if the agency which gives rise to the second sound was capable of communicating a distinct sensation to the hand or stethoscope.

In the healthy heart the second impulse is scarcely felt, unless the organ beats vigorously; when the ventricles are somewhat hypertrophied, and their cavities somewhat dilated, the second impulse becomes better marked; when this has arrived at an extreme degree, it becomes very evident, and constitutes, then, the "back stroke of the heart," or the diastolic impulse. This diastolic impulse, except in cases of disease, is never so strong as to be perceptible to the eye, but is readily distinguished when the ear is applied to the stethoscope laid upon the præcordial region. It is perceived at the same part of the chest as the systolic impulse, and is more marked the larger the surface of the heart uncovered by lung, and the stronger the action of the organ.

When describing the motions of the heart, we saw that during the ventricular diastole the apex of the heart recedes from its base, the organ becomes elongated, the ventricles increase in all their dimensions, and the hand, grasping the heart, is forcibly opened. Now, when we consider how closely the anterior surface of the ventricular portion of the heart lies to the parietes of the thorax, there is no difficulty in understanding how an impulse may be communicated during this movement equally as during the ventricular systole; it appears only surprising that it should have been so very generally overlooked.

## SOUNDS OF THE HEART.

If the ear, either with or without the stethoscope, is applied to the præcordial region in a healthy subject, two sounds are heard, which succeed each other quickly, but are readily distinguished from one another, and are followed by a pause or interval of repose; after which, the same phenomena are repeated. If the heart's action is very slow, a short but distinct interval may be re-

cognised between these two sounds, and the pause or interval of repose is longer; if, on the other hand, the action of the heart is very quick, the sounds follow one another with such rapidity that there scarcely appears to be any interval of repose.

The sound which occurs first in order after the pause is termed the first sound of the heart, sometimes the systolic sound; that which follows it is termed the second sound, sometimes the diastolic sound. The first of these sounds, compared with the second, has a dull character, and its duration is longer; the second sound is short and clear, and was compared by Laennec to the lapping of a dog, or to the falling back of a valve. The first sound is heard at the period of the ventricular systole, and for this reason has been named the systolic sound; it is synchronous with the impulse of the heart, and with the pulse. The second sound occurs at the period of the ventricular diastole, and for this reason is sometimes termed the diastolic sound: it immediately precedes the pause or interval of repose.

Both these sounds are readily distinguished at every part of the præcordial region: both are well marked at that part of this region where a dull sound is elicited by percussion; that is, where the heart approaches most closely to the parietes of the chest. Cruveilhier, in his examination of an infant born with the heart exposed, ascertained that both sounds increased in intensity from the apex towards the base of the heart, and that the maximum intensity of both sounds was at the same place, "hence (he observes) it is at the base of the organ we are to look for the cause of these sounds." Both sounds of the heart are usually well marked at the point where the apex is felt to beat, as well as over the base of the ventricles. The first sound has, however, its greatest intensity on a plane lower than the second; and M. Pigeaux, from this circumstance, proposed the name *inferior* sound for the first, and *superior* sound for the second. The sounds developed at the orifices of the right ventricle are better heard towards the right edge of the sternum; those developed at the orifices of the left ventricle are better heard towards the left edge of this bone; and the American experimenters determined that the ventricular systolic sound on the right side was sharper and clearer than that on the left, which was more dull and prolonged.

The double sound of the heart, Dr. Williams observes, may be

represented by the words *lubb*, *dŭp*. Dr. Walshe is of opinion that no *single* articulate symbol is applicable to the heart's sounds in more than one point, and he gives the following table to represent these sounds at different parts of the præcordial region :

	First Sound.	Second Sound.
At the left apex	übb	dŭp
At the right apex	úp	tŭp
At the left base	up	túp
At the right base	ub	tüpp

*Normal limits and intensity of the Heart's sounds.*—There is considerable difference, even in health, in different individuals in the intensity, as well as in the extent over which the sounds of the heart are audible : in some subjects they are limited to the præcordial region, and are comparatively feeble there ; in others, they extend much beyond the præcordial region, and are loud there. Whether the sounds are feeble or the contrary, and whether they are limited to the præcordial region or not, will depend upon a variety of circumstances—as the intensity of the sounds themselves, which is different in different subjects ; as the coverings of the chest are thick or thin ; as a larger or smaller surface of the heart comes in contact with the parietes ; as the lungs are largely developed and overlap the heart, or the contrary ; and, as the chest is capacious or narrow.

Dr. Latham,\* in his admirable lectures, speaking of the “natural limits of the heart's sounds,” observes—“It is a preliminary point which some have thought most important to be determined with precision ; but no good ever comes from pretending to more precision than the thing itself admits of—and I am sure this matter does not admit of much. The præcordial region, it has been said, defines it ; but surely the second sound always exceeds that limit, and is audible also in the course of the aorta, of the pulmonary artery, and of the carotids.” “With respect to the first sound, I should be at a loss to mark the exact space within which healthy proportion and healthy structure always required it to be heard—and in neither more nor less. There are so many circumstances—some consistent with health in the largest sense, and some exclusive at least of its disease—which make the systolic sound of the heart more or less extensively audible, that (I am persuaded) two healthy persons would not easily be found in

\* Lectures on Diseases of the Heart, vol. i.



whom it would be heard exactly within the same thoracic space. Whether a man be fat or lean will always make a great difference. In the one, it will be kept within the præcordial region ; in the other, it will be carried beyond it. Fat is so bad a conductor, that, where it greatly abounds, it will restrict the sounds to less than the entire præcordial region, even to a very small part of it ; so that you will not be able to hear the heart further than you can feel its impulse, or further than its apex—but mere skin and bone are such good conductors, that in very thin persons the sounds will be spread very far beyond the præcordial region, and will be heard at any part of the chest to which you apply your ear.”

#### MECHANISM BY WHICH THE SOUNDS OF THE HEART ARE PRODUCED.

We come now to the most difficult part of the subject—the cause of the sounds of the heart, or the mechanism by which they are produced : it is the one upon which the greatest difference of opinion has existed among physiologists and anatomists, and for the explanation of which the greatest number of hypotheses have been advanced.

*Theories of the cause of the first sound.*—It must be borne in mind, that at the period at which the first sound of the heart is heard,

1st. The muscular walls of the ventricles contract, and the cavity of the ventricles is diminished.

2ndly. The mitral and tricuspid valves suddenly pass from a state of relaxation to one of tension, and close their respective orifices.

3rdly. The blood is propelled with considerable force into the aorta and pulmonary artery.

4thly. The valves at these orifices are suddenly elevated ; and

5thly. The impulse of the apex of the heart against the parietes of the chest takes place.

Now, the first sound of the heart must obviously be due to one or other of these acts, or to a combination of them ; and it will be necessary to notice, shortly, the theories which have been advanced under this head.

As it has been completely proved that the first sound of the heart accompanies the ventricular systole, any theory founded upon a different order of succession of the heart's motions must be rejected ; such as, that it is due to the systole of the auricles,



or to the diastole of the ventricles, or to the systole of the left ventricle alone—the second sound being attributed to the systole of the right; because, it is well known, that the two ventricles contract simultaneously; that the ventricular diastole occurs at the period of the second not of the first sound; and that the auricular systole does not take place at the period of the first sound of the heart.

The theories which have been advanced in order to explain the mechanism by which the first sound of the heart is produced, may, for convenience sake, be considered as the cause is supposed to be extrinsic or intrinsic to the heart. Thus, under the first, it has been attributed to the impulse of the apex against the parietes of the chest; under the second head, it has been attributed to muscular contraction—in other words, to the successive shortening of the muscular fibres of the parietes of the ventricles. This is the oldest theory: it was adopted by Harvey, Haller, Senac, Bichât, and Corvisart. 2ndly. To the sudden tension of the auriculo-ventricular valves. 3rdly. To the friction of the blood against the parietes of the interior of the ventricles, or of the orifices of the large arteries. 4thly. To the collision of the opposite internal surfaces of the ventricles at the conclusion of the systole. 5thly. To the sudden elevation of the sigmoid and semi-lunar valves, caused by the wave of blood transmitted by the ventricles. 6thly. To the concussion of the blood transmitted by the systole of the left ventricle with that contained in the aorta; and lastly, to two or more of the foregoing causes combined.

*Theory of the sound being produced by the impulse.*—Majendie is the principal supporter of the theory, that the first sound of the heart is caused by the impulse communicated by the apex of the organ to the parietes of the thorax during the ventricular systole. M. Skoda likewise refers the first sound in part to this cause. It has, however, been distinctly heard in experiments upon animals, where the anterior parietes of the chest were removed and the heart fully exposed, as well as in infants labouring under ectopia cordis.

*Theory of muscular contraction.*—Those who refer the first sound of the heart to the muscular contraction of the walls of the ventricles include two phenomena under this head—viz., “the sound of muscular extension or tension,” and the “bruit muscu-

laire" or "rotatoire." Dr. Hope refers this sound essentially to muscular extension; which, according to him, is "a loud smart sound, produced by the abstract act of sudden jerking extension of the already braced muscular fibres at the moment that the auricular valves close." "It is (he observes) essentially different from the bruit musculaire, since it may be produced in a dead muscle, and may attain a high degree of loudness or sharpness; whereas bruit musculaire can only be produced in a living muscle, and is never more than dull and subdued." Other physiologists suppose the cause of this sound to lie exclusively in bruit musculaire. Dr. Blakiston is of opinion that it is caused by "the friction of the muscular fibres of the walls of the ventricles *inter se*."

*Theory of the collision of the opposite walls of the ventricles.*—By some physiologists, the first sound of the heart has been attributed to the collision of the opposed internal surfaces of the ventricles at the conclusion of the systole; but, as it is a doubtful point whether the ventricles completely empty themselves during their systole, and as it is quite certain that no such collision of the opposite walls of the ventricles takes place, this theory may be dismissed.

*Valvular theory.*—By other physiologists, the first sound of the heart has been attributed to the sudden tension of the mitral and tricuspid valves at the moment of the ventricular systole. Dr. Billing in England, and M. Rouanet in France, are the most distinguished advocates of this theory. But, as the auriculo-ventricular valves close their respective orifices at the very commencement of the systole of the ventricles, this act is certainly not capable of producing the prolonged first sound of the heart. According to M. Cruveilhier, on the other hand, the first sound is due, not to the closure of the mitral and tricuspid valves, but to the straightening or elevation of the sigmoid and semilunar valves, caused by the wave of blood transmitted by the ventricles.

*Theories of the motion of the blood.*—According to M. Pigeaux, the first sound of the heart is due to the friction of the blood against the walls of the ventricles, and the orifices and parietes of the large arteries, during the ventricular systole. According to M. Gendrin, it is due to the "vibrations which result from the sudden change of form which the blood experiences

during the ventricular systole." According to others, to the collision of the particles of the blood against each other, and against the walls of the ventricles, during the systole.

Dr. Leared,\* in a recent interesting communication upon the mechanism of the sounds of the heart, details some experiments which show, that a sound closely allied to the normal first sound of the heart can be produced "by the shock occurring between two portions of a liquid of a certain consistence, one of which, on being forcibly propelled by an intermittent action, is brought into contact with the other in a state of rest, or comparatively so." He gives the following explanation of the mechanism by which he supposes the first sound of the heart to be produced. "Subsequent to the elastic reaction of the aortic walls, which we must suppose does not occupy the entire period of the diastole of the ventricle, the column of blood in the upper part of the aorta attains a state of momentary repose. This column in a normal state is under considerable tension, and it is perfectly isolated from the contents of the ventricle by the semilunar valves—when the systole occurs, the valve with its superposed blood is forcibly thrown forward by the vigorous propulsion of blood from the ventricle; concussion now ensuing between the active and passive portions of blood, a sound is produced on the same principle, and from the same cause as in my experiments; and this *cæteris paribus*, is the essential element in the normal first sound of the heart."

*Theories of the cause of the second sound.*—The second sound of the heart is heard at the period of the ventricular diastole; and it must be borne in mind that during this act

1st. The muscular fibres of the walls of the ventricles are relaxed, and the ventricular cavities become enlarged.

2ndly. The auriculo-ventricular valves are opened, and the blood passes from the auricles into the ventricles, through the mitral and tricuspid orifices.

3rdly. The sigmoid and semilunar valves are pressed down by the column of blood above them, and the aortic and pulmonary orifices are closed.

The second sound of the heart must, therefore, have its cause in one of these acts, or in a combination of them; and any

\* Dublin Quarterly Journal, May, 1852.

theory founded on a different order of succession of the heart's motions cannot be correct—as that this sound is due to the auricular systole (Laennec), because the auricles contract immediately before the ventricles; or that it is due to the auricular diastole (Beau), because no sound is produced during this act; or that it is due to the systole of the right ventricle (Piorry)—because both ventricles contract simultaneously.

The theories which have been advanced to explain the mechanism by which the second sound of the heart is produced may, like those of the first sound, be considered under the head of causes extrinsic to the heart, and causes intrinsic to it. Thus Majendie attributes the second sound to the impulse communicated by the ventricles to the parietes of the chest during their diastole; Dr. Turner to the falling back of the heart against the pericardium at the moment of the ventricular diastole; and M. Skoda supposes it to be due, partly to the sudden disengagement of the apex of the heart from the opposed pericardium. But the second sound was heard in experiments upon animals when the ribs and sternum were removed and the pericardium laid open, as well as in infants labouring under ectopia cordis.

Those who refer the second sound to causes intrinsic to the heart, consider that, like the first sound, it may have its cause in muscular action, in valvular action, or in the motions of the blood. Thus, this sound has been supposed to be due, 1st, to the stretching of the muscular fibres of the ventricles in their diastole; but the muscular fibres are relaxed during this period of the heart's action. 2nd, to the rush of blood through the auriculo-ventricular orifices during the ventricular diastole. 3rd, to the shock of the blood against the parietes of the ventricles during their diastole. 4th, to the sudden closure or tension of the sigmoid and semilunar valves at the moment of the ventricular diastole. 5th, to the recoil of the column of blood in the aorta and pulmonary artery upon the sigmoid and semilunar valves at the moment that the ventricles dilate. 6th, to the “molecular collision of the blood during its recoil from the suddenly-closed semilunar valves.” The latter, Dr. Davies thinks, has been too much neglected in Skoda's theory of the second sound. Dr. Hughes\* compares it to “the recoil-noise heard when the closure of a stop-cock sud-

\* Clinical Introduction to Auscultation.

denly arrests the flow of water through a pipe." Dr. Leared considers that the second sound of the heart, equally with the first, is a concussion sound; here, however, the concussion is "between a fluid and a membranous expansion, instead of between two fluids."

*Theory of the sounds of the Heart most generally received.*—The theory of the mechanism by which the sounds of the heart are produced, which is most generally received, is as follows:—

*First sound.*—The first sound of the heart is regarded as a compound sound, partly valvular and partly muscular, the valvular portion being its first and loudest part, and being due to the sudden tension or closure of the mitral and tricuspid valves. The muscular portion of the sound, which is dull and prolonged, is supposed to be caused by the contraction of the muscular fibres of the walls of the ventricles, and to be due essentially, according to Dr. Hope, to "muscular extension," but receiving a "prolongation, and possibly an augmentation, from bruit musculaire;" according to others, to bruit musculaire alone, or to friction between the fibres of the muscular tissue of the ventricles.

That this sound is partly due to the sudden tension of the mitral and tricuspid valves, is considered to be proved "by the sound being loudest over the parts of the ventricles nearest to the auricular valves;" 2ndly, "when valvular extension was prevented by holding the mitral valve open (in experiments upon animals), this greatly diminished the first sound;" 3rdly, "whenever the auricular valves were destroyed, or the blood evacuated out of the ventricles, the sound became dull and obscure;" and lastly, by the character of the first sound in dilatation with attenuation of the ventricles, when it closely resembles the second sound.

That the first sound of the heart has its cause, likewise, in contraction of the muscular fibres of the walls of the ventricles, is considered to be proved by the "character of the sound"—by its continuing during the entire systole—by its being still heard, although weaker, in the heart of animals removed from the body—"by its being heard, although modified, in animals, when the auriculo-ventricular valves were prevented from acting, or when the blood was prevented from entering the cavity of the ventricles by pressing upon the orifices,"—"by the sound being louder

over the surface of the ventricles than over the origin of the large arteries,—and finally, by the sound being loud and short, when the walls of the ventricles are thin; and dull and prolonged when its walls are thick;” because a thick ventricle, *cæteris paribus*, takes a longer time to contract than a thin one.

*Second sound.*—The second sound of the heart is supposed to be due either to the sudden tension of the valves at the orifices of the aorta and pulmonary artery; or to the shock of the column of blood in the aorta and pulmonary artery, which recoils upon these valves at the moment of the ventricular diastole. This theory is supposed to be proved “by the second sound of the heart being loudest over the sigmoid and semilunar valves, and a little above them”—“by the sound ceasing in experiments made upon animals, when the reflux of the blood upon the semilunar valves was prevented by compressing the arterial orifices with the fingers”—and “by its being diminished when a semilunar valve in one artery was hooked up, and replaced by a murmur from regurgitation when the same was done in both arteries.”

The following experiment was made by M. Bouillaud, to prove that the second sound of the heart is caused by the reflux of the column of blood upon the sigmoid and semilunar valves. He attached one extremity of a short glass tube, of an inch bore to the aorta immediately *below* the semilunar valves, and to its other end a bladder full of water. Another tube four feet long, was connected with the aorta *above* the semilunar valves. The bladder was suddenly compressed at intervals, so as to jerk up the fluid, and each time that the pressure on the bladder ceased, and the column of liquid was allowed to fall back upon the valves, a sound very analogous to the second sound of the heart was heard.

A somewhat similar experiment was made by Dr. Corrigan, but with a different result. He removed the heart and ascending aorta of an ass, and then “tied it on the end of a leaden tube of a corresponding diameter, about five feet long; about two or three inches of the aorta then being free from the lower extremity of the tube. In this state, holding the sides of the aorta together below, he filled the tube with water, and then placing the thumb on the upper end, so as to close it, the fingers were withdrawn from the lower end, and the upper end still remaining closed, the external pressure of atmospheric air kept the two sides of the

aorta below together, and no fluid escaped. The ear was then applied to the lower end of the tube, close to the aorta, and the thumb being suddenly withdrawn from above, the whole column of fluid came suddenly down, and distended the aorta and valves; and yet there was no sound whatever similar to the second produced." He then "attached a piece of sounding-board, to assist the ear, and the result was the same as before."

*Remarks on the foregoing theory.*—The foregoing theory, it will be observed, omits from the elements capable of producing the sounds of the heart, all consideration of friction between the blood and the parietes of the orifices of the heart, in its passage into and out of the ventricles, during their systole and diastole. Yet when we come to describe the abnormal sounds heard in diseased states of the valves and orifices of the organ, we shall find that this very element, which is rejected as incapable of producing the normal sounds, is set down as the one which almost exclusively gives rise to the abnormal sounds of the heart. Now when we consider the force and rapidity with which the blood is propelled by the ventricles, particularly by the left, there can be no doubt that there is a considerable degree of friction between the blood and the parietes of the orifices through which it passes; and that this can scarcely happen without producing sound, appears evident, because even a slight impediment to the current of blood is sufficient to convert the normal first sound of the heart into a murmur.

Thus, in experiments made upon large animals, when the calibre of the aorta near its origin was narrowed, by pressing upon it during the ventricular systole, the first sound of the heart was converted into a murmur. Again, if the stethoscope is applied over a large artery, a short, slight, single sound is heard at each systole of the left ventricle; but, if pressure is made upon the vessel so as to diminish its calibre, a murmur will take the place of the normal sound. Again the blood in its passage through the veins causes no sound appreciable to the ear; under certain circumstances, however, sound is developed in particular veins, when their coats are made tense, and when their calibre is diminished by pressure with the stethoscope. This sound is familiar to us as the venous murmur.

Now, in each of these instances there is an obstacle or impe-



diment to the passage of the blood, and the increased friction which necessarily ensues is sufficient to convert the normal sound into a murmur. It seems probable, therefore, that abnormal sounds, are nothing more than exaggerated normal sounds, exaggerated because the friction between the blood and the parts through, or along which it passes, is increased; and if we admit, as the foregoing instances seem to prove, that normal sounds can be converted into abnormal sounds, simply by increase of friction, it seems not unreasonable to conclude that both are developed by the same agency.

In addition, we know that in aneurism springing from the arch of the aorta, a double sound is constantly audible, and this double sound remarkably resembles the double sound of the heart; so close indeed is the resemblance, that the second sound of aneurism in this situation is erroneously supposed by many to be the second sound of the heart transmitted to the aneurismal sac. We also know that the first, or the second, or both the aneurismal sounds are not unfrequently converted into murmurs, which have precisely the character of the murmurs heard in cases of valvular disease of the heart.

Now, in aneurism, in this situation, we have simply a sac communicating with a large artery by a single orifice, which is constantly patent; the sac is traversed by the blood propelled at each systole of the left ventricle, and into it the blood regurgitates at each diastole of the ventricle; yet every variety of normal and abnormal sound developed in the heart, with its muscular walls and valvular apparatus, may be produced also in an aneurismal sac which has neither the one or the other. It is scarcely, therefore, unreasonable to conclude, that the agent which is capable of generating sound in the one case is capable of developing analogous sounds in the other; and that, as sounds almost precisely similar to those of the heart, in its healthy as well as in its morbid state, can be produced independent of valvular extension, or muscular contraction, the latter are not such essential agents in the production of the heart's normal sounds as is generally supposed.

#### THEORY OF FRICTION BETWEEN THE BLOOD AND THE PARIETES OF THE ORIFICES OF THE HEART.

From what precedes, it would appear that sounds in every respect analogous to the normal sounds of the heart may be de-



veloped independent of valvular action, or of muscular contraction; while we know that the normal sounds of the heart are readily converted into murmurs, simply by increase of friction between the blood and the parietes of the orifices of the heart. Now, when we consider the rapidity and the force with which the blood enters and is expelled from the ventricles; and when we consider the amount of friction which must necessarily take place between this fluid and the parietes of the orifices of the heart, it seems not unreasonable to refer the normal sounds of the heart to this cause rather than to valvular action or muscular contraction: the first sound to the friction between the blood and the parietes of the arterial orifices during the ventricular systole; the second sound to the friction between the blood and the parietes of the auriculo-ventricular orifices during the ventricular diastole.

*First sound.*—The first sound of the heart we know is synchronous with the ventricular systole: in this act, the blood, compressed by the contraction of the powerful muscular walls of the ventricles, is propelled with considerable force into the aorta and pulmonary artery, the sigmoid and semilunar valves of which are suddenly elevated. In the rapid passage of the blood from a wider to a narrower area, there must be considerable friction between this fluid and the parietes of the arterial orifices; quite sufficient, in my mind, to produce the prolonged first sound of the heart. This sound necessarily has a distinct character from the second sound of the heart, because the resistance to be overcome is so much greater, and the passage of the blood through these orifices is more gradual; it is likewise more prolonged, because sound must be developed during the entire period that the blood is passing from the ventricles into the large arteries: and the slower the action of the heart, the more prolonged will this sound be.

*Second sound.*—The second sound of the heart we know is synchronous with the ventricular diastole: during this act the muscular fibres of the ventricles are relaxed, the cavity of the ventricles enlarges, and the walls of the ventricles re-expand; the curtains of the auriculo-ventricular valves open, and there is a sudden influx of blood from the auricles through the auriculo-ventricular orifices. It is scarcely necessary to say, that it is not the contraction of the auricles which propels the blood into the

ventricles at this period of the heart's action ; nor is the dilatation of the cavities of the ventricles the result of the entrance of blood from the auricles, as some have supposed. It is not, either necessary for the production of this sound that the diastole of the ventricles should be an active process like the systole ; the ventricles being hollow muscles, the state of relaxation of their muscular fibres is a state of dilatation of their cavities : hence a vacuum would be created in them if the auricles were not at this period full of blood ready to supply them, but, as the latter had been filling during the whole period of the ventricular systole, this cannot happen, and the blood passes through the auriculo-ventricular orifices in a full and rapid stream, and with sufficient force to generate sound.

That the blood enters the ventricles with considerable force would appear from what has been observed in experiments upon animals, as well as in the human subject. Cruveilhier says that in a case of ectopia of the heart in an infant, when the organ was grasped with the hand during the ventricular diastole, it was violently and forcibly opened,—so much so, that he was at first under the impression that the diastole was the active state of the ventricles.

The second sound of the heart is much shorter than the first sound, because the relaxation of the muscular fibres of the ventricles in the diastole is rapid, and the motion is sudden and instantaneous. It has a different character from the first sound, because the blood has no impediment to overcome in entering the ventricles from the auricles.

*Objections to the theory of valvular tension as a cause of the first sound.*—It is said that the heart, being a muscular organ, and its orifices being each provided with a distinct valvular apparatus, the contraction of the muscular tissue of the ventricles or the action of its valves, ought to be capable of producing sound. Now, the first sound of the heart cannot have its cause exclusively in the sudden tension of the auriculo-ventricular valve because this act takes place at the very commencement of the systole ; and the first sound is a dull, prolonged sound, which persists during the entire systole ; again, it occasionally happens that the mitral valve is rendered rigid and immovable by disease without any murmur from regurgitation occurring, yet the first

sound is still perfectly well marked, which could not be the case if it was due to valvular tension. That any part of this sound is produced by valvular action seems to be doubtful, because the curtains of the mitral and tricuspid valves are floated towards the orifices by the blood which distends the ventricles; and, when the ventricular systole commences, the auriculo-ventricular orifices are mechanically closed by the pressure of the blood against the curtains of these valves.

*Objections to the theory of muscular contraction as a cause of the first sound.*—It remains, therefore, to consider how far the first sound is due to muscular contraction. This is supposed to be proved by the first sound having been heard in experiments upon animals when the organ was removed from the body and continued to contract, because there was then neither collision of the blood nor valvular action; but when a heart contracts and dilates under such circumstances, air must enter the ventricles during their diastole, and be expelled during their systole, and the friction of the air against the parietes of the orifices of the heart, is quite as capable of developing sound, as the friction of a fluid.

Three theories, of the manner in which sound is produced by the contraction of the muscular fibres of the ventricles of the heart, have been proposed. Dr. Hope, as I have already observed, refers it to “muscular extension;” Dr. Blakiston,\* to friction between the muscular fibres themselves during the act of contraction; and others to “bruit musculaire.”

According to Dr. Hope, the sound of muscular extension is “a loud, smart sound, produced by the abstract act of sudden jerking extension of the already braced muscular walls, at the moment when the auricular valves close.” With respect to this theory of the production of sound, I must confess I do not exactly understand how sound can be produced in this way, at all resembling the first sound of the heart.

By many physiologists the first sound of the heart has been referred to “bruit musculaire,” which, according to Mr. Bowman,† is “an exceedingly faint, silvery vibration.” The mechanism by which bruit musculaire is produced, may (he thinks) be explained

\* Practical Observations on Diseases of the Chest.

† Philosophical Transactions.

“by supposing the several fasciculi to be in rapid and constant motion, one against the other, by varying amounts of contraction in different fasciculi and parts of fasciculi.” Dr. Blakiston’s theory of friction between the muscular fibres themselves, and Mr Bowman’s theory of the mechanism of *bruit musculaire*, seem therefore, to be pretty nearly the same.

Laennec\* was the first to call attention to *bruit musculaire* in connexion with the sounds of the heart. In the part of his work where he endeavours to explain the mechanism by which *bruit de soufflet* is produced, he observes: “When the naked ear or the stethoscope is applied over a muscle in a state of contraction, or better still, upon one extremity of the bone to which the muscle is attached, we hear a sound analogous to that of the wheels of a carriage at a distance, and which, though continuous, is evidently formed by a succession of very short and quickly succeeding sounds; or if, with the head resting upon a pretty firm pillow the masseter muscles are strongly contracted, and then contracted with less force; in the former instance the wheel seems to roll with great rapidity upon a hard surface, in the latter it seems to roll over a rough pavement.”

Dr. Williams,† who refers the prolongation of the first sound of the heart to *bruit musculaire*, observes: “Whenever there is strong, abrupt, muscular action in any part of the body, like that of the heart, there will be heard a sound which will resemble that of the ventricular systole, in proportion as the muscles, in which it is produced, resemble in thickness and density the tissue of the heart; as when we apply the stethoscope to the adductor muscle of the thumb of the closed hand, and contract the muscle strongly and quickly; or, to avoid the possibility of the joints being the seat of the sound, if we apply the end of a flexible tube to the abdominal muscles, and start them into sudden vigorous action we may thus get sounds quite as loud as those of the ventricles and very like them in character. By varying the mode of this muscular action, different kinds of sound may be produced. When the contraction is slow or sustained, however strong, we have only the dull rumbling noise which Dr. Wollaston described, and which he attributed to a vibration depending on a regular intermittence in the force of the contraction. When the contrac-

\* *Traité de l’Auscultation Méd.* tome ii.

† *On Diseases of the Chest.*

tion is gentle and slow, it may cause no sound at all; as we have seen that the auricles produce no sound, neither do the ventricles when their contraction is very feeble."

Dr. Watson,\* who likewise adopts the theory of bruit musculaire, observes, in reference to the first sound of the heart: "If during the stillness of the night, when lying in bed, you set the teeth firmly, you will hear a continuous dull rumbling, caused evidently by the action of the masseter and the temporal muscles."

Now, the dull rumbling sound which is heard under such circumstances, if it is to be taken as the type of bruit musculaire, is very unlike the first sound of the healthy heart. It is a continuous, dull, rumbling noise, and has a much greater resemblance to the venous murmur heard in the jugular veins in cases of anemia than to it. Indeed, I am convinced that this sound has not its seat in the masseter or temporal muscles, for it is heard only in the ear which rests against the pillow, not in the opposite ear. The ear must likewise be pressed pretty firmly against the pillow, by which the air is confined between the tympanum and the external ear. Indeed, a sound altogether similar will be heard if the ear is stopped with the finger, and the temporal and masseter muscles are then strongly contracted. The sound has certainly nothing of the "faint silvery vibration" which, according to Mr. Bowman, characterises bruit musculaire; and the theory which refers the prolonged portion of the first sound of the heart to it, appears to me to rest upon too hypothetical grounds to entitle it to be considered as the cause of this sound, or of any portion of it.

*Objections to the Theory of the Recoil of the Blood upon the Arterial Valves as a cause of the Second Sound.*—The second sound of the heart is generally supposed to have its cause in the sudden tension of the sigmoid and semilunar valves; or in the recoil of the column of blood contained in the aorta upon these valves, or in both combined.

That these valves close their respective orifices at the period in question, we know; but that their sudden tension, or the recoil of the column of blood upon them is the cause of the second sound, I have considerable doubts, thus:

1. The closure of these valves is described by some as a

\* Lectures on the Practice of Medicine.

sudden flapping motion. But these valves do not flap against one another, and it cannot be against the blood which then begins to enter the ventricles by the auriculo-ventricular orifices, because the arrangement of the tricuspid and mitral valves is such, that the blood does not reach them at this period.

2. The instant the onward current from the ventricles ceases, the blood in the aorta insinuates itself into the little sinuses behind these valves, and by its simple weight presses them down and closes the orifices.

3. The amount of force with which a column of blood should fall upon them, in order to develop this sound, would necessarily soon cause these valves to be reversed; and, if they were exposed to such a rude shock at every diastole of the ventricles, few persons could attain adult age in whom they would not permit regurgitation.

Those who adopt the foregoing theory, of the mode of production of the second sound of the heart, seem to argue as if the arteries were inelastic tubes, and that the blood in the aorta and pulmonary artery had a backward as well as a forward motion. If this were so, it could hardly fail to be perceived in the large arteries which come off from the arch of the aorta: while, for a column of blood to come in contact with these valves with sufficient force to develop the second sound of the heart, presupposes an empty space in the artery above them, into which the blood falls back at each ventricular diastole. But as long as the artery preserves its elasticity, nothing of the kind can occur; the blood does not recoil with any force, or from any distance, upon these valves, because the artery immediately above them is filled equally with the other parts of the tube. In fact, the instant that the onward current from the ventricles ceases, the blood by its simple weight depresses these valves, and as long as the parts are healthy there is no recoil of a column of blood upon them. But when, as occasionally happens, the arch of the aorta loses its elasticity, and becomes dilated, there is then certainly a recoil of the blood contained in it upon the semilunar valves; and this state almost always eventually ends in patency of the aortic valves.

*Pathological evidence in favour of the Theory of Friction, between the Blood and the parietes of the Orifices, as a cause of the Sounds.*—It will now be necessary to adduce some pathological evidence that the sounds of the heart are caused by friction

between the blood, and the parietes of the orifices of the ventricles, during their systole and diastole. I have already said, that a double sound, which cannot be distinguished from the normal double sound of the heart, is heard in cases of aneurism of the arch of the aorta; and that in certain instances the first of these sounds, in others the second, and in others again both these sounds, are converted into murmurs, altogether analogous to the murmurs which accompany diseased states of the orifices and valves of the heart. That the aneurismal sounds are caused by friction between the blood, and the parietes of the orifice of the sac, requires no proof, because there is no other agency to which they could be referred. This is at least evidence that a valvular apparatus and muscular walls are not essential to the production of sounds analogous to those of the heart. The conditions common to both are, a cavity into and out of which the blood passes with more or less force and rapidity; and if the friction between the blood and the parietes of the mouth of the sac of an aneurism is capable of developing, not merely a double sound similar to that of the heart, but murmurs which differ in nothing from those developed in diseased states of the heart, the same agent is evidently equal to produce analogous sounds at the orifices of the ventricles.

*First sound.*—If the first sound of the heart has its cause in the friction between the blood and the parietes of the arterial orifices; and if murmurs are nothing more, in the great majority of cases, than exaggerated normal sounds, then the normal first sound of the heart ought to be converted into a murmur, whenever an impediment is offered to its exit; when the blood is propelled with increased force and velocity, or when the qualities of the blood are altered, and this fluid loses its viscosity and becomes more watery than natural; because in these several cases the friction between the blood and the parietes of the orifice is increased. The sound ought to diminish in intensity, or in duration, when less blood is transmitted by the ventricle, or when it is transmitted with less force; and it ought to be more prolonged than natural when an increased quantity of blood is transmitted by the ventricle during its systole: and this is exactly what does occur. For instance:

1. Whenever the aortic orifice or its valves become diseased so as to *obstruct* the outward current of blood, the normal first



sound of the heart is converted into a murmur, which will have either a blowing, or harsh character, according to the amount of the obstruction, and to the force and velocity of the current.

2. When the walls of the left ventricle are hypertrophied, and the blood is propelled through the aortic orifice with increased *force*, although there is no impediment to its exit, the normal first sound may be converted into a murmur. In the ordinary explanation of the mechanism by which the first sound is produced, a murmur from this cause ought not to occur.

3. When the viscosity of the blood is diminished, and this fluid is propelled with increased *velocity* through the aortic orifice, the normal first sound is converted into a murmur.

4. When the walls of the left ventricle are attenuated, the first sound of the heart comes to resemble the second sound, because the blood is propelled with less force, less blood is transmitted through the arterial orifice, and the systole lasts a shorter time.

5. When the walls of the left ventricle are softened, or have undergone fatty degeneration, the first sound of the heart becomes more feeble, because the blood is propelled into the aorta with less force; and, when the aortic orifice or the semilunar valves are diseased so as to obstruct the outward current from the ventricle, no abnormal sound will be heard, because the force of the current in such cases is too feeble to convert the first sound of the heart into a murmur.

6. When the cavity of the left ventricle is dilated, and its walls are increased in thickness, a larger amount of blood will be transmitted at each ventricular systole, and the first sound of the organ will necessarily be prolonged.

It may be objected to the foregoing theory, that the abnormal sound does not always replace the normal first sound of the heart, and that the latter is still heard in some cases of valvular disease along with it. But as valvular disease is almost limited to the left side of the heart, the abnormal sounds are limited in the same proportion to the left side; and, if a normal sound is audible along with the abnormal, it may be the normal sound of the right side of the organ, which the murmur was not sufficiently intense to mask completely.

*Second sound.*—If the second sound of the heart has its cause in friction between the blood and the auriculo-ventricular orifices,



it ought to diminish in intensity whenever there is an impediment to the entrance of the blood from the auricle, or when the ventricle remains distended, owing to its inability to empty itself. This sound is very seldom converted into a murmur, because there is nothing to increase the force with which the blood enters the ventricles from the auricles, though there are several which may diminish the force or velocity of the current from the auricles into the ventricles. For instance :

1. When the mitral orifice is much contracted, the second sound of the heart will diminish in intensity ; and, when the contraction is extreme, it is scarcely audible, because but little blood can enter the ventricle from the auricle during the ventricular diastole.

2. When the ventricles of the heart of animals, submitted to experiments, were gorged with blood the second sound was scarcely heard, or ceased, because a sufficiently strong current of blood could not enter the ventricle to develop sound. This was noticed both in the experiments performed in the United States and in England. "The second sound (Drs. Pennock and Moore observe), by the congestion of the ventricles ceasing first on the right side." "When the heart was gorged, towards the conclusion of the experiments, the first sound (Dr. Hope observes) only was heard."

3. Although the second sound itself is seldom converted into a murmur, a murmur which masks the second sound is by no means rare, and is heard whenever the aortic valves permit regurgitation. Why a murmur should be heard in this case, or why the second sound of the heart should be seldom converted into a murmur, is easily understood. When the mitral orifice is contracted, less blood can enter the ventricle during its diastole, and the second sound becomes more feeble than natural : it is not converted into murmur, because the force with which the blood enters the ventricle from the auricle is too slight to produce this effect. On the other hand, when the aortic valves permit regurgitation, as these valves are on a higher plane than the auriculo-ventricular orifice, and as it is the blood contained in the aorta above these valves which regurgitates into the ventricle, the amount of friction between the blood and the abnormal orifice is sufficient to generate a murmur in this case, though not in the

former ; but, the force with which the blood regurgitates into the ventricle never being so great as that with which it is expelled from the ventricle, the murmur of aortic regurgitation is usually soft and blowing, and seldom acquires the rough, harsh character which a systolic murmur often presents.

In addition, we know, that when patency of the aortic valves exists, two currents of blood must enter the ventricle at each diastole ; that through the aortic orifice being the stronger, for the reasons already given, a smaller amount of blood will therefore necessarily enter by the auriculo-ventricular orifice ; and the force with which it enters will be diminished, because the ventricular cavity is partially filled by the backward current through the aortic orifice. Hence, we have another reason why the normal second sound should become more feeble than natural, and why it should be so readily masked by the louder sound of aortic regurgitation.

Lastly, the occurrence of intermission of the heart's sounds, or irregularity of the heart's action, which are with difficulty explained on the theory of the heart's sounds ordinarily received, admit of a ready explanation on this. For instance :

If too little blood is expelled by the left ventricle at each systole to communicate a pulse to the radial artery, and if this occurs at every second, third, or fourth beat, as the case may be, there will necessarily be an intermission of the pulse ; while, if the amount of blood is too small, or the force with which it is propelled too slight to occasion friction at these periods between the blood and the parietes of the orifice, the heart's sounds themselves will be intermittent.

If the left ventricle becomes overloaded with blood, or its parietes are weak, the systole of the ventricle may be repeated several times for one diastole ; and this may occur at short intervals, so that the pulse may be felt to give several rapid and small beats, and then several stronger and more regular beats, as if it were the pulse of two different persons ; and the same irregularity will be perceived in the sounds of the heart when the stethoscope is laid on the præcordial region.

These phenomena, which scarcely admit of explanation according to the ordinary theory of the heart's sounds, admit of a ready explanation if we allow that the sounds are due to

friction between the blood and the parietes of the orifices of the ventricles; that if little blood is transmitted, or it is transmitted with little force, little sound can be produced; while, when the force and velocity of the circulation are increased, the friction will be increased in proportion; and, when an impediment is offered to the passage of the blood under such circumstances, the normal sound will be converted into a murmur.

## CHAPTER V.

EXAMINATION OF THE HEART IN DISEASE.—SIGNS FURNISHED BY INSPECTION OF THE CHEST, AND THE APPLICATION OF THE HAND.—SIGNS FURNISHED BY PERCUSSION.

THE anatomy and physiology of the healthy heart having been dwelt upon at sufficient length, we have next to consider the heart in disease, and to describe the several methods of proceeding, in order to determine the nature, the situation, and the extent of the morbid changes. And here we shall find the advantage of the preceding details, inasmuch as we shall be in a position to recognise readily any alteration in the heart's impulse, either as respects its strength, or the situation in which it is felt; we shall be able to detect any difference in the extent and degree of the heart's superficial dulness, or any change in the character of its sounds.

In cases where the heart is diseased, or suspected to be so, several distinct methods of examination require to be employed; the eye, the touch, the ear, are each capable of affording us assistance in arriving at a diagnosis, and the indications afforded by each mutually assist the others. These methods of examination may, for convenience sake, be considered under the heads—inspection of the chest; palpation, or the application of the hand to the parietes; percussion and auscultation—upon each of which it will now be necessary to delay for a short time.

*Inspection of the Chest.*—In other words, the examination of the external surface of the thorax, and the application of the hand to the præcordial region should never be omitted; they are capable, alone, of affording positive information in several diseased conditions; they aid and assist our other means of arriving at a diagnosis; and, as preliminary steps to other and more difficult methods of exploration, they can scarcely be dispensed with, if we wish to make an accurate examination.

As these two methods of physical examination mutually assist

one another, they may be considered together. Thus, by inspection of the chest, we determine the exact point at which the apex of the heart comes in contact with the parietes of the chest, and by the application of the hand we determine the strength or feebleness of its impulse. By inspection of the chest we ascertain whether the two sides of the thorax are symmetrical, and we detect any protrusion in the præcordial region; by it we recognise unusual pulsation at any part of its parietes, in the large arteries which come off from the arch of the aorta, as well as in the jugular veins, or in the epigastric region. By the application of the hand we determine the force of the pulsation, we judge of the frequency or slowness of the heart's action, and of the regularity or irregularity of its movements. By the application of the hand, likewise, we recognise the peculiar phenomenon known under the name of *fremissement cataire*, or purring tremor; and by the same means we can detect friction between the opposed and roughened surfaces of the pericardium in inflamed states of this membrane.

*Impulse of the Heart.*—The application of the hand to the præcordial region is necessary, in order to judge of the strength or feebleness of the heart's impulse: with this view, the hand may either be placed directly upon the surface of the chest, or, holding the ear-end of the stethoscope, we apply its opposite extremity to the part, when the extent to which the instrument is elevated, and the force with which this is accomplished, will give us a pretty accurate idea of the strength of the heart's impulse. This kind of mediate palpation is principally useful in hypertrophy, or hypertrophy with dilatation of the ventricles.

The impulse of the heart varies, even in healthy individuals, according to the development of the chest, according to the size of the lungs, according to the fatness or leanness of the individual, according to the stature of the subject, and to the size of the abdomen. In the healthy subject, with a well-formed chest, the impulse of the heart is so slight as not to be perceptible to the individual himself, and is *felt* only at one point—viz. between the cartilages of the fifth and sixth ribs on the left side; that is, from one to two inches below the nipple, and to the sternal side of this point. When the parietes of the chest are much loaded with fat, the impulse is scarcely perceptible to the hand; while in thin persons it is evident also to the eye. In such cases, when the heart's

action is vigorous, it will be perceived to be double; and this in certain states of disease becomes very perceptible.

In examining the heart, it is necessary to bear in mind that the impulse is somewhat stronger in the erect than in the recumbent posture; that in a forced inspiration the impulse is diminished, and felt slightly lower down than natural; a deep inspiration elevating the ribs without raising the heart in the same degree, while it depresses the diaphragm. That in a forced expiration the impulse becomes more perceptible, and is felt slightly higher up; a forced expiration depressing the ribs and elevating the diaphragm. It may therefore be necessary to make the patient vary his position, and to examine the heart both during inspiration and expiration. In some individuals the impulse is naturally weak; in others, naturally strong: the same may be said of the pulse and of the sounds of the heart, although the parts are in a perfectly normal condition in each. As the impulse is increased by exercise, or exertion, or by mental excitement, we should be careful to examine the heart suspected to be diseased when the patient is perfectly calm and tranquil.

In some morbid conditions of the heart or lungs the impulse is either very slight or altogether absent. In others, it is so strong as to be disagreeable to the individual, and to be visible without removing the patient's clothes, and the extent of surface over which it is felt is much increased. In others, the impulse becomes perceptibly and strongly double; in others, the seat of the impulse is altered, or it becomes irregular in rhythm and force; or, finally, it may be altogether absent.

*Diminution of the Impulse.*—Diminution of the impulse of the heart may depend, either upon feebleness of the action of the heart, which may have its cause in disease or alteration of its muscular tissue, or in general debility of the system; or it may depend upon the apex of the organ being prevented from coming in contact with the parietes of the chest, with sufficient force to communicate an impulse, owing to disease in the lungs or pericardium.

The impulse of the heart is circumscribed or feeble in softening of the heart, and in fatty degeneration of its tissue. In states of considerable general debility, it is feeble. The impulse is diminished, likewise, in attenuation of the walls of the ventricles, with dilatation of their cavity. If the anterior margin of both lungs is

emphysematous, and they overlap the heart, the apex may be prevented from coming in contact with the parietes, and the impulse will be much diminished. In cases of considerable effusion into the sac of the pericardium, the impulse is absent altogether, or it is unequal and undulating, as if propagated through a fluid. In cases even of hypertrophy with dilatation of the ventricles, the impulse may be diminished, "it becomes a mere oppressed struggle," if the heart is overloaded with blood and the lungs are much congested.

*Increase of the Impulse.*—Increase of the impulse of the heart, as a general rule, occurs under the very opposite conditions of the organ to those under which diminution of its impulse is observed, and, in the majority of cases, it has its cause in some morbid state of the heart itself. The impulse is stronger than natural in hypertrophy of the walls of the left ventricle, and it arrives at its utmost limit in hypertrophy with dilatation of the ventricles; in such cases the impulse is slow, gradual, heaving, double, and occasionally so violent as to shake the bed on which the patient lies. The character of the impulse of the heart thus becomes a most valuable sign of hypertrophy with dilatation; "the slow, progressive, heaving impulse could be produced by no other cause." In such cases, likewise, the extent of surface over which the impulse of the heart is felt, is much increased; and the whole side of the chest is sometimes elevated by the movements of the organ. It is in this form of disease that the double impulse is so well marked, the diastole as well as the systole of the ventricles being accompanied by an impulse: this is sometimes termed the back stroke of the heart, more correctly the diastolic impulse. The ventricles may, however, be considerably hypertrophied, and the heart much enlarged, without any increase of the impulse. Thus in a case, recently under my care, where the heart, upon a post-mortem examination weighed 28 ounces, the impulse was not increased, owing to the pleuræ of opposite sides having contracted adhesions with one another, and with the outer surface of the pericardial sac; by which the apex of the heart was prevented from coming in contact with the parietes. M. Piorry says, that in aged females at the Salpêtrière very little impulse is often felt, although the heart is frequently much thickened; while in nervous subjects with small hearts the impulse is frequently very strong. He is of opinion

that increased impulse is to be considered “an indication rather of the force of the blow than of the thickness of the parietes of the ventricles.”

In hypertrophy with a predominance of dilatation, the impulse, as Laennec observes, is abrupt, short, and knocking, and in the fits of palpitation the shock resembles the blow of a hammer. “The blow seems (as Dr. Hope remarks) to strike a small space: it expends itself, as it were, on the thoracic parietes, and does not communicate a heaving proportionate to its force.” “It differs from the impulse of great hypertrophy, in the circumstance that in the latter the ventricles, in a distended state, seem to heave with their whole length against the thoracic parietes, which yield to the effort; while, in the former, the point only of the heart seems to strike the parietes with a sharp, smart, accurately circumscribed blow, only capable of producing a sort of concussion, rather than a real heaving.”

The impulse of the heart is stronger than natural in the early stage of endocarditis, and of pericarditis; likewise, in attacks of palpitation from any cause; and it is apparently stronger when a morbid growth behind the heart protrudes the organ forwards, as in certain rare cases of aneurism in the descending portion of the thoracic aorta. The impulse becomes sharper and more knocking in nervous and hysterical subjects, particularly during fits of palpitation: in such cases it is often very troublesome to the patient, and is occasionally accompanied by perceptible sound.

*Alteration in the situation of the Impulse.*—The situation at which the impulse of the heart is felt is not unfrequently altered by disease, either of the heart itself, of the lungs, or pleura, by the development of morbid growths in the cavity of the thorax, or by morbid or other changes in the contents of the cavity of the abdomen; and, these alterations in the site of the impulse, may be the result either of the increased volume which the heart itself attains, or of displacement of the entire organ. Thus:

In hypertrophy of the left ventricle with dilatation of its cavity, the heart being increased in length, its impulse will be felt lower down than natural, more to the left side, and, occasionally, on a line with the axilla. In hypertrophy with dilatation of the right ventricle, the impulse will, for the same reason, be felt lower down and more to the right side than natural, and, not unfrequently, on



a line with the xyphoid cartilage. In the former case the impulse is progressive, heaving, and strong, elevating the head of the observer, and is felt over a much larger surface than natural; in the latter the impulse is neither heaving, prolonged, or very strong, and is felt over a more circumscribed space.

The entire organ is displaced laterally in cases of empyema, towards the left side when the right pleural cavity is its seat, and towards the right side when the left pleural cavity is its seat: in the latter case the impulse is frequently felt upon the right side of the sternum. The heart is pushed upwards, and the impulse is felt on a plane higher than natural in ascites, in cases of large ovarian, or other abdominal tumors, in hysterical tympanitis, and the advanced stages of pregnancy. The heart is displaced slightly downwards in cases of emphysema of both lungs of long standing, and the impulse is felt on a plane lower than natural, not unfrequently in the epigastric region. In cases of effusion of fluid into the pericardial sac, the site of the impulse is somewhat elevated; when the amount of fluid is greater, the impulse becomes weaker, unequal, undulatory, or irregular; when the amount of liquid effusion is very considerable, the impulse will be altogether absent. When the parietes of the ventricles have undergone fatty degeneration, or when they are softened, or much thinned, the impulse is generally irregular. In individuals with narrow, deformed, or contracted chests, the impulse will be perceived beyond its normal limits. In individuals, on the other hand, with broad and expanded chests, the site of the impulse is usually circumscribed.

*Metallic Cliquetis.*—Although under ordinary circumstances, sound is not produced by the impulse of the heart, and no shock or blow is given to the parietes of the chest by the apex of the organ during the ventricular systole—the intercostal space between the cartilages of the fifth and sixth left ribs being merely elevated by the apex of the heart in the gliding movement performed by it at this period of the heart's action—yet, under certain circumstances, sound is really produced by this act, which in certain rare cases is sufficiently loud to be heard without the stethoscope, and at a short distance from the patient, or is audible to the patient himself.

Corvisart only once witnessed this phenomenon. Laennec states that in at least twenty instances which he met, this sound

was sufficiently loud to be audible at a distance of from two inches to two feet from the patient's chest. "In only three or four of these cases, at the utmost, organic disease of the heart existed; in all the others, the palpitation was purely nervous. Bruit de soufflet and fremitus often existed in a slight degree in the heart, but, particularly in the arteries of those who presented this phenomenon."

In order that this sound should be produced, the heart must act strongly, the subject must be thin, the heart must be a good deal uncovered by lung, and the apex of the organ must come in contact with the rib or its cartilage with sufficient force to produce sound.

According to Dr. Hope, the manner in which the sound is produced is as follows: "The heart in gliding forwards and upwards during its systole strikes with its apex against the *inferior margin* of the fifth rib, and thus creates an accidental sound, attended with *cliquetis* when the blow is smart. It may be prevented at pleasure, by pressing the edge of the stethoscope, or any thing else, into the intercostal space, by which that space is put internally on the same plane as the rib, over which the heart then glides without catching." He adds, that, "he has never found the sound to occur in any but the meagre, because in the well-conditioned the intercostal spaces are full and resistant, and consequently the edge of the rib is not exposed."

In order to hear the metallic click, M. Bouillaud says it is better not to employ a stethoscope, "this sound being sometimes heard with the naked ear when it would be inaudible with the stethoscope"—Dr. Ormerod,\* who has investigated this phenomenon with much care, has met with it under four different conditions—viz., 1st, in nervous, anæmic subjects, when the action of the heart was sharp; 2ndly, with more violent action of the heart; 3rdly, at the commencement of pericarditis; and 4thly, with a rough, almost scaly state of the pericardium about the base of the heart; to which he thinks may be added adhesion of the pericardium. He says "there are three explanations of the mode of production of this sound—namely, costal percussion, friction of free surfaces, and movement of connecting areolar tissue; each probably applies to a limited number of cases—

\* Gulstonian Lectures, *Medical Gazette*.

there does not appear to be any one general explanation, nor considering the nature of the sound, ought we, perhaps, to look for one."

*Turgescence and pulsation of the jugular veins.*—Among the signs which are evident on inspection of the chest, and which accompany the advanced stage of some forms of cardiac disease, a state of permanent turgescence or distension of the jugular veins, with or without pulsation, is by no means unfrequent. The former is the most common, and may occur in any case in which an impediment exists to the free passage of the blood through the right side of the heart. The latter is observed in cases in which, in addition, the tricuspid valve imperfectly closes the right auriculo-ventricular orifice, and free regurgitation into the auricle is permitted at each systole of the right ventricle.

Turgescence and pulsation of the jugular veins were first noticed, by Lancisi, as signs of cardiac disease. He referred them to dilatation with hypertrophy of the ventricles, or, as it was then termed, "aneurism of the heart." Corvisart had little confidence in them as signs of disease of the right side of the heart. Laennec, however, regarded the turgescence without pulsation of these veins as a frequent attendant upon dilatation of the right cavities of the heart, and he says that "pulsation in the same veins was present in every case which he had met with of considerable hypertrophy of the right ventricle."

The internal and external jugular veins receive the blood from the interior and exterior of the head, from the face and neck. Now, if there is any impediment to the free passage of this fluid through the right side of the heart, whether depending upon disease in the right or left side of the organ, or of the lungs, these veins become distended and turgid. If, in addition, the tricuspid valve does not close the right auriculo-ventricular orifice, a portion of the blood, at each systole of the right ventricle, instead of passing into the pulmonary artery, will be transmitted backwards into the right auricle, and will re-act upon its contents, and through it upon the blood descending by the superior cava and its branches, when a pulsation will be communicated to the jugular veins at each systole of the right ventricle, which will persist although pressure be made upon the vein above it.

The diseased condition of the heart in which turgescence of the jugular veins is most frequently observed, is dilatation of the right auricle and ventricle, with or without hypertrophy of the same parts. It is, however, by no means to be considered a symptom belonging exclusively to this form of disease, for it is often the result of obstruction, or regurgitant disease at the left side of the heart. It may accompany any impediment to the free circulation of the blood through the cavities of either side of the heart. It is observed sometimes in aneurism of the ascending part of the aorta, as well as in diseased conditions of the lungs, accompanied by obstruction to the pulmonary circulation. The turgescence is usually limited at first to the external jugular veins, particularly on the right side: it is not constant, and can always be removed by compressing the vein above. Eventually both the internal and external jugular veins become distended and turgid, sometimes to an extreme degree. This state is permanent, though it can still be removed by pressure upon the vein higher up. In some few cases, the veins, in addition to being dilated, become tortuous, and present the appearance with which we are familiar in the varicose state of the veins of the lower extremity.

The veins in which pulsation is observed are the internal jugulars, those vessels not being provided with valves like the external. The pulsation is always most evident immediately above the clavicles, and it may extend half-way up the neck when the latter is short; but I have never found it to reach higher. It is usually best marked on the right side, and is frequently perceived upon both sides, and sometimes, though rarely, only on the left side. The pulsation is too feeble in the great majority of cases to be felt by the finger, but, in several instances which have come under my observation, a pulsation was communicated to the finger placed lightly upon it. This could not be confounded with the pulsation of the artery underneath, which is likewise generally increased at the same time—as it was very feeble, was perceptible only on slight pressure, and disappeared when this was increased—when the impulse of the artery alone was felt.

The movement in jugular pulsation is rapid, vermicular, and double; the backward current of blood forming the first and

strongest movement, and the direct current its second and slighter movement. According to Dr. Sibson, there is in the healthy state "a constant visible pulsation, both in the deep and superficial jugular veins, which pulsation, though perfectly visible, cannot be felt." It is much diminished, and in many persons rendered invisible, during a deep inspiration. "The mere existence of jugular pulsation (Dr. Sibson observes) is anything but an indication of disease, either in the pulmonary valves, or elsewhere." "In those diseases (he adds) where the flow of blood through the lungs and heart is impeded, the jugular veins contain more blood, and their pulsations are more visible than in health; but where the impediment is extreme, the veins are in a state of constant distension, and no pulsation is visible."

Pulsation of the jugular veins is present in every case in which the tricuspid valve imperfectly closes the right auriculo-ventricular orifice, and free regurgitation occurs at each ventricular systole, provided the walls of the right ventricle are sufficiently strong to communicate a backward impulse to the current of blood which descends by the jugular veins. Hence it is almost always observed in hypertrophy with dilatation of the right cavities of the heart. Pressure upon the vessel between the site of the pulsation and the heart is always sufficient to stop it.

The cause of jugular pulsation was first satisfactorily explained by MM. Bertin and Bouillaud—viz., that it depends upon regurgitation into the great veins during the contraction of the right ventricle; and this theory of its cause has been almost universally adopted. Dr. Hope, however, supposed that it might have its cause in the recoil of the tricuspid valve upon the blood, when the hypertrophied ventricle contracted with increased force, and that the column of blood descending into the ventricle would be repelled with sufficient force to propagate its impulse as far back as the jugular veins. From the explanation which has been given of the manner in which the curtains of the auriculo-ventricular valves close their respective orifices, it would appear that jugular pulsation could scarcely be produced in this way. Why regurgitation is permitted at the tricuspid orifice so frequently, as well as the exact manner in which it occurs, have been already alluded to in treating of the "safety-valve function of the tricuspid valve."

## FREMISSEMENT CATAIRE.

Another sign which is recognised only by the application of the hand is the “fremissement cataire” of Laennec, the “purring tremor” of English writers, the “frottement fremissement ou vibratile” of Gendrin; named so by Laennec, from its resemblance to the peculiar sensation experienced if the hand is laid upon the back of a cat when the animal is making the peculiar purring sound which it does when pleased; and compared by M. Bouillaud to the thrill felt on placing the hand upon the larynx of a person singing. It is a peculiar thrill, or vibratory sensation, very easily recognised, sometimes perceptible on very slight pressure sometimes requiring a stronger pressure in order to be felt, and communicated equally by the large arteries as by the heart.

This phenomenon had been recognised as a sign of cardiac disease previous to the discovery of auscultation. Corvisart, speaking of the symptoms of disease of the left auriculo-ventricular orifice, observes—“Among the symptoms of this affection there is a peculiar confused sensation, difficult to describe, felt by the hand applied to the præcordial region.” He supposed it to indicate a contracted state of the mitral orifice, causing an impediment to the passage of the blood from the left auricle into the left ventricle. Burns evidently alludes to this phenomenon when he speaks of a “jarring motion accompanying the pulsation of the heart;” and, as he noticed it in several cases where, on a post-mortem examination, the opposed surfaces of the pericardium were adherent, he attributed it to this lesion.

Fremissement cataire is not limited to the heart, but is perceptible also in the large arteries, and in aneurism, particularly in varicose aneurism, in which it is better marked than in the heart. It is felt over the large arteries when the lining membrane of these vessels is extensively diseased, as well as in cases where no morbid alteration of the parts exists; and its cause, or the mechanism by which it is produced, do not seem to have ever been fully explained. Laennec\* observes—“It would seem that the immediate cause of a phenomenon so marked as fremissement cataire would be easily discovered. I confess, however, that, notwithstanding the pains which I have taken in this re-

\* Auscultation Mediate, tom. ii.

spect, I have not been able to find any satisfactory reason." "It appears to me (he adds) extremely probable that it is owing to some peculiar modification of innervation." M. Bouillaud,\* in the second edition of his work on the heart, expresses himself in somewhat similar terms: "What are the other causes capable of producing this phenomenon, I am ignorant. It appears to me that there is a certain condition of the blood, as well as of the parietes of the heart and arteries, which favours its production. But in spite of the assiduous researches to which I have devoted myself on the subject, I have not yet been able to determine properly all these conditions."

*Circumstances under which Fremissement Cataire occurs.*—In every instance in which fremissement cataire is perceived, a murmur of some kind is audible on auscultation, which is generally blowing, sometimes rough or harsh. It is obvious, therefore, that the cause which produces the latter is connected with that which gives rise to the former; but, as fremissement cataire is not felt in *every* case in which an abnormal sound is heard, it is obvious that there must be something superadded when the latter is felt to allow of this peculiar sensation being communicated to the hand.

The morbid condition of the heart in which this phenomenon is most generally perceptible is where the left auriculo-ventricular valve permits regurgitation, and the ventricle is at the same time hypertrophied and dilated; it has occasionally also been noticed in cases of disease of the aortic valves with considerable contraction of the orifice, but this is rare. The morbid conditions in which it is felt in the arteries, are where the aortic valves permit free regurgitation, or where the arch of the aorta is dilated, its lining membrane rough and irregular from adventitious deposit, and the large vessels which come off from its arch are likewise dilated; where an aneurismal sac springs from a large artery, where a free communication exists between a vein and artery, as in varicose aneurism; or, finally, where the blood is altered, its viscosity diminished, its watery parts increased, and the vessels are in an unfilled state.

Two or more of the foregoing states may be combined, by which the thrill will be increased. Thus dilatation of the arch of the aorta may be combined with disease of its lining membrane, and both may be associated with a state of the aortic valves per-

\* *Traité des Maladies du Cœur*, tom. i.



mitting regurgitation ; or dilatation of the large arteries, or regurgitant disease of the valves of the heart, may be combined with a state of anæmia and alteration of the blood.

*Cause of Fremissement Cataire.*—In every case in which fremissement cataire is felt, whether in the heart, or in the artery or in aneurism, the cavity or the vessel is in an *unfilled* state. Thus in the heart it becomes perceptible when the mitral valve permits regurgitation ; in the large arteries, when the aortic valve is patent, when the arteries themselves are dilated, when aneurism exists, or when a communication exists between an aneurism and artery. Now I consider this unfilled state of the vessels, combined with a certain amount of force and velocity of the current of blood, to be the immediate cause of this phenomenon : the tremor or vibration communicated to the hand being more marked when the lining membrane of the part is rough or irregular from disease.

For instance, we can produce a fremissement at any time in a large artery in a healthy subject by making pressure upon it ; the thrill is not felt at the point upon which the pressure is made, but between it and the heart, but at the distal side of the point of pressure, and where the vessel must necessarily be in an unfilled state : a murmur is heard at the point compressed, and both cease as soon as the pressure is removed. We know, too, that a thrill identical with fremissement cataire, is felt in cases where the heart and arteries are perfectly healthy, but where the vessels are in an unfilled state, as in anæmia, depending upon hæmorrhage or other causes.

A certain amount of force and velocity of the current of blood appears to be likewise necessary to its full development ; because we find that this phenomenon diminishes or even disappears in diseased states of the heart, when the action of the organ becomes tranquil, and that it reappears again when the heart's action is excited by exercise. It disappears likewise when the action of the heart becomes feeble ; and ceases altogether in the last stage of valvular disease, when the circulation through the heart is completely impeded.

The jarring pulse in the radial artery, with which we are so familiar in cases of patency of the aortic valves, appears to be nothing more than the fremissement cataire felt in an artery of small calibre. The thrill communicated to the finger is chara-



istic of an unfilled state of the arteries, and becomes perceptible whenever the semilunar valves of the aorta imperfectly close the orifice, or when regurgitation occurs into the aorta from the large arteries which come off from its arch. The same character of the pulse is observed in cases where profuse hæmorrhage has occurred; and in a less marked degree in cases of anæmia from other causes.

Fremissement cataire is or is not a sign of organic disease according to its situation; thus, whenever it is felt in the præcordial region, accompanies the ventricular systole, and is well marked, it indicates valvular disease; and the form of valvular disease which will be found under such circumstances is, in the great majority of cases, a state of the mitral valve or orifice permitting regurgitation. In the large arteries, on the other hand, a fremissement may be felt, although no organic disease of the vessel exists. In some instances, this phenomenon is perceptible when the hand is laid gently upon the parietes: in others, stronger pressure is required in order to develop it. Thus in varicose aneurism, a thrill is communicated to the finger on the slightest touch; while in cases of anæmia some pressure upon the artery is required in order to make it evident.

A peculiar thrill or vibration is occasionally perceptible in the region of the heart, which must not be confounded with the phenomenon just described, and which arises from the direct friction of two rough surfaces during the motions of the heart: here it depends upon friction between the opposed layers of the pericardium, roughened by the deposition of lymph; the thrill has a different character from that of fremissement cataire, and is felt at a different part of the præcordial region. A somewhat similar feel is occasionally experienced when the hand is laid upon the side in cases of pleuritis or pleuro-pneumonia with false membranes; in bronchitis, likewise, when the sonorous râle is loud, a sensible vibration is sometimes communicated to the hand: in the latter cases, however, it accompanies the respiratory movements, not the heart's action.

The præcordial region includes the whole of that portion of the chest beneath which the heart is situated; and, in a healthy subject with a well-formed chest, the extent of surface in this

region which yields a dull sound on percussion is limited. We have seen that the lungs are in contact with the greater portion of the anterior surface of the thorax, and, of course, wherever they are, the sound elicited by percussion will be clear. Where the thin margin of the opposite lungs separates from one another, the anterior surface of the heart comes in contact with the thoracic walls, being only separated by the pericardium and cellular tissue.

The portion of the heart uncovered by lung is very small, seldom exceeding two inches in any direction: it has a triangular shape—the base below, the apex above; it consists of a portion of the apex of the right ventricle, and of part of the left ventricle near its apex, and is seated on a plane below the nipple and the fourth ribs; its base is on a line with the cartilage of the sixth rib; its apex is at the point where the margins of the opposite lungs begin to separate from one another, viz., immediately below the fourth rib; its right boundary (which is constituted by the thin edge of the right lung) is nearly a vertical line through the centre of the sternum; its left boundary (which is constituted by the thin margin of the left lung) is an oblique line through the cartilages of the fifth and sixth left ribs.

This, then, is the only portion of the heart which, in the healthy subject, is in contact with the parietes of the chest, and it is the only portion of the præcordial region which yields what can be termed, a dull sound on percussion. It is not, however, quite correct to term this a dull sound; it is less dull than that yielded by the hepatic region: the liver being a solid organ, and the heart hollow, the sound elicited by percussion over the former is much duller than over the latter. Indeed, as Dr. Latham\* observes, “percussion here conveys to the ear a sense rather of less resonance than of positive dulness.” On stronger percussion, a difference in the sound can be detected where the thin margin of the lungs covers the heart. The sound here is intermediate between the clear sound heard on percussion over the lungs, and that yielded by the part of the præcordial region where the heart is in contact with the parietes of the chest.

In employing percussion in cases of disease, we may commence the examination of the præcordial region either above, below, or laterally, we may trace the sounds yielded by the lungs above and upon each side to this region; or, commencing below, we may

\* Lectures on Diseases of the Heart, vol. i.

trace the sounds from the region of the stomach, and of the left lobe of the liver, upwards. The mode of manipulation recommended by Dr. Hope\* is “to lay one finger over the decidedly dull part, and another over the slightly resonant edge of the lung, when by striking the two fingers alternately, the arched line along which the organ lies in contact with the walls may be traced with surprising accuracy, unless the subject be remarkable for obesity, which obscures the resonance.” “In females, the mamma may be pushed upwards, which generally leaves the dull portion sufficiently accessible.”

*Auscultatory Percussion.*—Another means of estimating the size of the heart has been termed by Drs. Camman and Clark, its discoverers, auscultatory percussion†—“a solid cedar cylinder, six inches in length, and one inch in diameter, cut in the direction of the fibres, and with an ear-piece attached, is applied to the centre of the præcordial region while the ear is applied to the other end, percussion is then made by another person from the point near where the cylinder is applied towards the limits of the heart in every direction.” “So long as percussion is made over the body of the heart, a distinct sharp shock is felt directly in the ear; but, as soon as the limits of the heart are passed this sharp shock immediately ceases, and that even in passing from one solid organ to another in contact with it, as from the heart to the liver.” “Care must be taken not to confound this shock with that more diffused shock produced by striking the ribs; a little practice will render the discrimination easy.” “To make a practical illustration of this method, apply the solid stethoscope over the centre of the præcordia, and percuss upward just outside the origin of the aorta until the sharp shock abruptly ceases—mark the spot, then percuss downward until the shock again ceases—mark this spot also; connect the two points by a vertical line; this will give the vertical diameter, and so with the rest.” By this mode of measurement, the mean diameter of the healthy adult heart was found to be as follows, in inches and lines—

	Male.				Female.	
Vertical diameter	.	.	4	0 lines	3	7 lines.
Transverse	.	.	4	4	4	1
Right oblique	.	.	4	10	4	10
Left oblique	.	.	3	10	3	7

\* Treatise on Diseases of the Heart, 4th edition.

† Swett's Treatise on Diseases of the Chest.

*Alterations in the extent and degree of the Heart's superficial dulness.*—The extent of surface in the præcordial region which yields a duller sound than natural on percussion, is seldom diminished, owing to diseased states of the heart; but it is very often enlarged, while the degree of dulness is not unfrequently at the same time increased. The space will be diminished if the heart is congenitally smaller than natural, or if the lungs are largely developed; and it will disappear if the anterior margin of both lungs is emphysematous, and their edges meet in front of the organ. As the heart, however, is generally enlarged under the latter circumstances, a portion of its anterior surface will still come in contact with the parietes, but the space which yields the dull sound will be on a plane somewhat lower than natural.

The region of the heart's superficial dulness will be increased whenever the heart is enlarged, or whenever fluid to any amount is effused into the sac of the pericardium. Thus, if the walls of the ventricles are hypertrophied, or if their cavities are dilated, a dull sound will be elicited by percussion over a wider surface, and the extent of this surface will be, in some degree, a measure of the increased size which the organ has attained: the enlarged heart pushing aside the lungs, and a larger portion of it coming in contact with the parietes. When the two foregoing conditions are combined, the heart attains the largest size that it is capable of, and the præcordial region may yield a dull sound over a square surface of from two to six inches. When the hypertrophy predominates over the dilatation, the space which yields a dull sound is wider from above downwards; when dilatation predominates over the hypertrophy, the region in which a dull sound is yielded is wider transversely. In pericardial effusion a larger surface than natural in the præcordial region yields a dull sound, while the degree of dulness is more pronounced, and the sensation of resistance is considerably greater than in the former case.

When a dull sound is elicited by percussion over an extensive surface in the præcordial region, the situation in which it is most marked, and the amount or degree of dulness, will be a guide to its cause. When it depends upon enlargement of the heart, the site of the dulness is lower down, and more to the left side than when it depends upon liquid effused into the sac of the pericardium. As the pericardial sac extends upwards to a level with

the articulation of the second rib on the left side with the sternum, and sometimes, as I have found to be the case, as high as the first rib, if a large amount of fluid is contained in this sac a dull sound will be elicited by percussion as high as this point; while the degree of dulness on percussion over a fluid is always much more marked than over the heart itself, and the resistance to the finger is greater. In addition, the history of the case, and the rapidity with which the other symptoms have set in, will generally enable us to distinguish enlargement of the heart from pericardial effusion. It is said that hypertrophy may be distinguished from simple dilatation of the ventricles by the degree of resistance being greater in the former than the latter; but there are other and better signs by which these conditions of the heart may be distinguished.

The results furnished by percussion are, however, sometimes doubtful; for instance, when the margins of the lungs are emphysematous, and approach each other in the præcordial region, a clear instead of a dull sound will be yielded in the natural situation of the heart's superficial dulness. When the lungs are solidified, either as the result of pneumonia or of tubercular infiltration, or from any other cause, a dull sound will be yielded upon each side of the heart over a considerable surface. When the heart is displaced, owing to liquid effusion into either pleura, or to some morbid growth in the cavity of the thorax, the region of the heart's superficial dulness will be altered.

*Conclusions respecting percussion of the Præcordial Region:*

1. The region of the heart's *superficial* dulness, in the healthy adult subject, does not exceed two inches in any direction.
2. This space has a triangular shape, the apex immediately below the fourth ribs, its base on a line with the cartilage of the sixth ribs; its right margin a nearly vertical line through the centre of the sternum, its left margin an oblique line from the fourth sterno-costal articulation through the cartilages of the fifth and sixth left ribs.
3. The sound elicited by percussion here is not absolutely dull except about the centre of this space.
4. The region of the heart's *deep-seated* dulness in the healthy adult subject, extends transversely from the left nipple to a little to the right of the sternum, and vertically from the third to the sixth ribs.

5. The region of the heart's *superficial* dulness may be diminished, or it may be increased, or the degree of dulness may become more marked, owing to disease. It is much more frequently increased than diminished.

6. It will be diminished if the heart is atrophied or congenitally small, or the lungs are large and overlap the organ; and it may disappear if the anterior margin of both lungs is emphysematous.

7. It will be increased whenever the ventricles are hypertrophied, or their cavities are dilated, or when fluid is contained in the pericardial sac.

8. The region of the heart's *deep-seated* dulness will be increased if the pulmonary tissue around is condensed or solidified or, from the presence of a morbid deposit in this situation; and it may be altered by liquid effusion into either pleura, or by the growth of intro-thoracic tumours.

9. The seat of the diminished resonance extends higher up and is more extensive from above downwards, and laterally, when much fluid is contained in the sac of the pericardium, than when hypertrophy or dilatation of the heart exists.

10. The degree of dulness is always much more pronounced and the resistance to the finger is greater when fluid is contained in the sac of the pericardium, than when the heart is enlarged.

## CHAPTER VI.

**SIGNS FURNISHED BY AUSCULTATION.—ALTERATIONS OF THE NORMAL SOUNDS OF THE HEART.—ABNORMAL SOUNDS.—PERICARDIAL AND ENDOCARDIAL MURMURS.—ARTERIAL AND VENOUS MURMURS.**

**AUSCULTATION**, like percussion, may be either immediate or mediate. Although immediate auscultation is preferred by some, there are several objections to it. Thus, in the case of females it is indelicate; in dirty persons it is disagreeable; while in contagious diseases it is not without risk: besides, there are some situations in which either the ear cannot be applied, or in which the stethoscope is much more convenient. In examining the precordial region in cases of valvular disease, or where there is a suspicion of it, mediate is always to be preferred to immediate auscultation: indeed, the exact situation, or the limits of an abnormal sound, can in the majority of cases be satisfactorily determined only by the assistance of the stethoscope.

As the sounds heard in a healthy state of the heart must be the standard of comparison in judging abnormal sounds, it is necessary to be familiar with them before commencing the examination of cases of disease: yet this is a matter too frequently overlooked by the student. He begins by examining the cases of disease which come under his observation in hospital, without, perhaps, any previous knowledge of the normal sounds. Now it is scarcely necessary to say, that, unless the character, duration, intensity, and other qualities of the normal sounds of the heart are known, the student is not in a condition to appreciate the numerous modifications or alterations which these sounds undergo in disease. It would, in fact, be almost as absurd to commence the study of anatomy by entering at once on pathology, without any knowledge of the healthy appearance of the organs, as to commence the practice of auscultation by examining cases of disease.

In cases where the heart is diseased, or suspected to be so, it is often advisable, before concluding the examination, to make the patient walk quickly up and down stairs, or backward and forward in the room, with the view of accelerating the circulation and increasing the heart's action—by which, abnormal sounds will be often rendered more evident, or a murmur which was inaudible previously may become distinct. Thus, a musical murmur is sometimes audible, only when the action of the heart is increased by exercise, a simple bruit de soufflet replacing it as the circulation becomes tranquil. When a murmur, which was inaudible as long as the circulation was tranquil, becomes evident when the heart's action is increased, it is highly probable that there is, as Dr Latham\* observes, “a mechanical obstacle at an orifice of the heart, but that it is of small amount, not enough to cause the requisite degree of vibration when the current of the blood is slow and undisturbed, but quite enough when it is more rapid and forcible.”

Again, in some cases it is necessary to examine the patient in the sitting or erect as well as in the recumbent position, or to make him lean forwards. Thus, in a case of pericarditis, under my care in hospital, the friction-sound at one period became nearly inaudible in the recumbent posture, although sufficiently evident in the sitting posture, and still more so when the patient leaned forward. Or the friction-sound may diminish in intensity in the sitting posture, and become well marked in the recumbent position obviously depending on the difference of the situation in which lymph is deposited. It may, in addition, be sometimes necessary to make the patient hold his breath, for a few seconds, during the examination, particularly if a murmur is not well marked or the sonorous or sibilant râles of bronchitis are loud. Lastly, if an difficulty should be experienced in distinguishing between the first and the second sound, or in determining to which a murmur should be referred, the finger ought to be kept on the radial pulse or, better still, upon the carotid artery, during the examination and the stethoscope should be applied on the right side of the sternum, and the sounds of the right cavities traced towards the left side.

Lacnec has made the remark, that when a portion of lung

\* Lectures on Diseases of the Heart, vol. i.



interposed between an enlarged heart and the parietes of the chest, the impulse of the organ, by compressing the lung and expelling the air from it, may give rise to an abnormal sound resembling some of those heard in disease of the heart. “A strong impulse of the heart on a portion of lung may (Dr. Williams\* observes) forcibly press the air from it; and, if there happen to be any partial obstruction or mucus in its tubes, a short sibilant or mucous rhoncus may accompany each beat.” “The character of these additions, and the circumstance that they accompany the breathing also, and are more or less diminished by holding the breath, or by posture altering the manner in which the heart beats on the lung, may serve to distinguish them from the true cardiac sounds.”

Again, it sometimes happens that, in the advanced stage of phthisis, each impulse of the heart is accompanied by a tinkling sound, resembling the tintement metallique. This occurs in cases in which a large tubercular cavity, containing a certain amount of fluid, occupies the greater portion of the upper lobe of the left lung, its lower lobe being at the same time solidified by tubercular deposition, and the action of the heart strong. The sound here may accompany both the impulse of the heart and the respiratory movements. In a case which was in hospital some time since (where this abnormal sound was sufficiently loud to be audible without applying the stethoscope, and at some distance from the patient's bed), it accompanied only the impulse of the heart, not the respiration, during a portion of the time that the patient was under observation.

In diseased states of the heart, its valves, or orifices, the sounds are variously modified or altered. Thus, in some instances, their intensity is greater or less than natural; in others their character or tone is altered; and in others their duration is affected. Sometimes the extent of surface over which they are heard is increased or diminished, or they may become more frequent, sometimes less frequent than natural; or their rhythm may be variously disturbed. Lastly, and in a very numerous class of cases, new sounds are superadded which either accompany the normal sounds or take their place.

*Alterations in the intensity and tone of the Sounds of the Heart.*—The intensity of the heart's sounds varies in different in-

\* On Diseases of the Chest.

dividuals, and under different circumstances: varieties are likewise met with in the character of its sounds, some of which are observed in cases of disease, while others occur independently of any morbid change in the heart. Thus, in persons in whom the parietes of the thorax are loaded with much fat, or where these parts are œdematous—in muscular subjects in whom the chest is largely developed, and whose lungs are ample, the sounds of the heart have less intensity than in individuals in whom the chest is narrow or deformed, in whom its coverings are thin, or in whom the cartilages of the ribs are ossified. Whenever, likewise, the energy of the heart's action is increased, the sounds as a general rule become loud. Thus, in nervous and hysterical subjects; in palpitation from any cause—under the influence of mental emotion, or after exercise—its sounds become more intense than natural. On the other hand, when the energy of the heart's action is lessened from any cause, the intensity of the sounds will be diminished.

The intensity or the tone of the sounds of the heart is frequently modified by disease; but this applies more particularly to the first than to the second sound: thus, when the walls of the ventricles are hypertrophied, the first sound becomes duller than natural; when the cavity of the ventricles is dilated, the first sound becomes clearer than natural; when the walls of the ventricles are attenuated, combined with dilatation of their cavities, the first sound somewhat resembles the normal second sound, while the second sound is feeble; when the muscular tissue of the heart is flabby or softened, both sounds become more feeble and obscure, besides undergoing other changes afterwards to be mentioned. In the advanced stage of fever accompanied by considerable debility, the sounds of the heart sometimes become extremely feeble during the state of syncope, also, its sounds are indistinct, and the second sound is often inaudible. In dilatation combined with some hypertrophy of the ventricles, both sounds are loud; when, however, the hypertrophy predominates over the dilatation, and this diseased state has arrived at an extreme degree, both sounds, but particularly the first, become obscure.

Clearness of the first sound of the heart is very generally considered to be an indication of dilatation of the ventricles; this, however, must be taken with some limitation, for this character of

the first sound is observed when neither dilatation or hypertrophy are present; thus, if the stomach is much distended with flatus the sounds might become clearer and louder than natural. Dr. Clendinning\* says he has found the first sound of the heart to be abnormally short, shrill, and clear, approaching the character of the second in numberless instances, in hearts proved post-mortem to be much and even enormously hypertrophied. According to him, "a short, clear, systolic sound indicates not so much any particular anatomical state of the heart, but rather a defective dynamic condition; that it indicates, in a word, not *attenuation* of the parietes, but merely *debility*." M. Piorry, likewise, attributes little importance to difference in the clearness of tone of the sounds, as a sign of hypertrophy, or dilatation of the ventricles. He says, he has "frequently found the parietes thickened when the sound was clear, and vice versa, and the tone frequently varies in the same person if subject to palpitation." He attributes clearness of tone to the amount of blood contained in the cavity, the sound being clearest when the heart is most empty, yet is contracting with energy, as in palpitation.

*Alterations in the duration of the Sounds of the Heart.*—The sounds of the heart are modified or altered in their duration, as well as in their intensity, in some diseased states of the organ. Thus, in hypertrophy of the walls of the ventricles, the first sound is more prolonged than natural; in dilatation of the ventricles, the first sound is shorter than natural. "The transition of a thick muscle from slack to tight can never be so complete and sudden (Dr. Williams† observes) as that of a thin one; where there are many fibres, they cloak and muffle each other's vibrations, hence the first sound is dull and prolonged; a thin ventricle, for the same reason, will give a louder and sharper sound." But if a thick ventricle takes a longer time to contract upon its contents than a thin ventricle, the blood must obviously pass out of the cavity more slowly in the former than in the latter; and as sound appears to be produced during the entire period that the blood is being transmitted from the ventricle, the first sound must necessarily be prolonged in this diseased state.

*Alterations in the limits within which the Sounds of the Heart are audible.*—The extent of surface over which the sounds of the

\* Croonian Lectures, *Medical Gazette*, vol. xxv. † On Diseases of the Chest.

heart are audible on auscultation, in the healthy and well-formed chest, is not great, being pretty nearly limited to the præcordial region ; and as the stethoscope is moved from this part, they become more and more indistinct. There is, however, considerable difference in this respect in different subjects ; in individuals whose chest is covered with much fat, and in whom the impulse is slight or hardly perceptible, the sounds are more limited ; while in thin subjects, in children and females, in individuals whose chest is contracted or deformed, and in nervous subjects, the sounds are audible over a wider area. The sounds produced at the right side of the heart are more perceptible upon the right side of the præcordial region, while those produced in the left cavities are more distinct at the left side of this region. When the sternum is short the sounds of the heart are usually audible in the epigastrium region.

In several diseased states of the heart or lungs, the sounds become audible over the greater part of the anterior surface of the chest, and sometimes, in addition, in the lateral and posterior regions upon both sides : while in other morbid states the sounds are very obscure, even in the præcordial region. Whenever the intensity of the sounds is increased, or whenever the heart itself is enlarged, its sounds, as might be expected, are audible beyond their normal limits, provided the circulation through the heart and lungs continues free ; hence in dilatation of the ventricles, and dilatation combined with hypertrophy, the sounds are widely diffused. In palpitation from any cause, particularly in anæmic, nervous, and hysterical individuals, the sounds of the heart will be heard over great part of the anterior surface of the chest, particularly if the subject is emaciated.

The sounds of the heart, however, frequently become audible on auscultation beyond their normal limits, even over the greater portion of the chest, independent of any alteration of the heart. This happens whenever the density of the pulmonary tissue is increased, by which its power of conducting sound is augmented ; hence, in solidification of the lungs from hepatization, from tubercular deposition, or from any other cause, the sounds of the heart will be audible to a considerable distance beyond their normal limits, and this very extension of the heart's sounds becomes a useful sign in these diseases of the lungs. In displacement of

heart the situation at which the sounds are heard will be of course altered ; but, as the seat of the impulse is likewise changed in such cases, it need not be dwelt upon here. A large amount of fluid in the pericardium might prevent the sounds of the heart from reaching the ear ; but there are other signs by which this diseased state may be more readily diagnosed.

*Alterations in the frequency of the Sounds of the Heart.*—

The sounds of the heart may be more frequent than natural, or they may become slower than natural, or they may be intermittent or irregular. They are more frequent than natural in all inflammatory affections of the heart or its coverings, as well as in numerous diseases of other parts, in states of debility, in cases of anæmia, and generally whenever palpitation arises, whatever be its cause. The heart's sounds much less frequently become slower than natural ; when they do, the cause lies more frequently in disease of the nervous centres than of the heart. Fatty degeneration of the heart appears to be the only diseased state ever accompanied by diminution in the frequency of the heart's action. In hypertrophy of the ventricles the heart's action is said to be sometimes slower than natural, it is not, however, an ordinary occurrence, because, although the first sound is more prolonged than natural, the interval of repose is usually shortened in proportion. Dr. Willis\* observes that, when considerable contraction of the aortic orifice exists, the pulse becomes much slower than natural: "here the ventricle, engaged in squeezing its charge through a hole little or no larger than a goose-quill, perhaps, has been found contracting with no greater frequency than twenty-five, twenty, and even sixteen times in a minute." It is not easy to understand how contraction of the aortic orifice can render the action of the heart slow, although it must, no doubt, considerably diminish the amount of blood transmitted at each systole of the ventricle.

*Alterations in the Rhythm of the Heart.*—In all the cases which we have been hitherto considering, whether the sounds of the heart were increased or diminished in intensity, whether they were more frequent or slower than natural, the rhythm of the organ was regular ; the two sounds succeeded one another, and were followed by an interval of repose, which varied in length according to the duration of the previous systole, and according to

\* Medical Gazette, vol. i. 1842—43.

the rapidity with which the sounds succeeded each other. It not unfrequently happens, however, that after a certain number of perfectly regular beats, a sudden pause or silence occurs; the heart's action and sounds appear to be arrested or suspended for a moment and then go on regularly, as before, when, after a certain number of beats, the same phenomenon is repeated. This constitutes intermittence or intermission of the heart's action; and it may occur after every fourth, eighth, or tenth beat, or at longer or irregular intervals. It is occasionally observed in individuals who are otherwise in perfect health: but it also occurs in diseased states of the valves or orifices of the heart, where either some impediment exists to the direct passage of the blood, or where regurgitation is permitted.

In the ordinary theory of the mechanism of the heart's sounds intermission is with difficulty explained; but if it be admitted that the sounds of the heart have their cause in friction between the blood and the parietes of the orifices of the heart, we can easily understand how, in certain diseased states, so little blood may be expelled from the left ventricle, or it may be propelled with so little force, that there is not friction sufficient to generate a sound, or force enough to produce an impulse, or to communicate a pulse to the radial artery. We know that the circulation may go on when the sounds of the heart are scarcely audible, and when no impulse can be felt in the præcordial region, as in syncope; yet, in such cases, the valves must perform their functions, and the muscular walls of the ventricles contract and dilate.

It sometimes happens that every systole of the left ventricle is not sufficiently strong, or the amount of blood transmitted by the left ventricle is not sufficiently great to communicate an impulse to the radial artery, and the pulse intermits: but on applying the stethoscope to the præcordial region, the sounds are still heard, although more feebly, and there is no real intermission of the heart's action. If this should occur at every second ventricular systole the pulse will appear to be preter-naturally slow: and this probably is the true explanation of some of the cases of slow pulse which have been recorded. The error would have been corrected by laying the stethoscope on the præcordial region.

In certain diseased states of the muscular tissue of the heart, of its valves, or orifices, the rhythm of the heart's sounds undergoes

Other alterations, the relative duration of the sounds to the intervals of repose is altered, and the latter may be increased or diminished. Alterations in the post-systolic silence are not easily recognised, unless the heart's action is slow; alterations in the post-diastolic silence, or the natural period of repose of the heart, are more frequent, and more easily distinguished. A more frequent alteration, however, consists in *irregularity* of the rhythm, the double sound of the heart being rapidly repeated twice or thrice, followed by two or three slower repetitions of the same sounds, some of these being strong, others weak, particularly the second sound which is sometimes imperceptible. This is observed in cases where the circulation through the heart or lungs, or through both, is impeded, and occurs in the advanced stage of several diseases of the heart, as in considerable contraction of the mitral orifice, in softening of the muscular tissue of the ventricles, in pericarditis with copious liquid effusion; or where fibrinous concretions are developed in the cavities of the heart previous to death. It is of course a much more unfavourable symptom than simple intermission.

*Alterations in the number of the Heart's Sounds.*—Another alteration in the rhythm of the heart's sounds, occasionally observed in cases of disease, is where, instead of the ordinary double sound, we have a triple sound, each systolic sound being repeated twice for one diastolic, or each diastolic sound being repeated more than once for one systolic sound. In other cases both the systolic and diastolic sounds are doubled, and four sounds are heard for one impulse of the heart. Lastly, it sometimes happens that only one sound is audible, in place of the ordinary double sound, and it is then always the second sound which is absent.

When we hear a triple sound in place of the ordinary double sound of the heart, it is more frequently owing to a reduplication of the systolic than of the diastolic sound. This reduplication of the first sound is compared by M. Bouillaud to the tattoo of the drum: it also resembles what is called in Ireland "the Kentish fire." When the diastolic sound is doubled, the triple sound closely resembles the footsteps of a cantering horse heard at some distance, to which Dr. Williams compared it.

This reduplication of the heart's sounds is seldom heard except in cases of disease, and, in many instances, some abnormal sound is audible at the same time. Sometimes this phenomenon is only



observed when the heart's action is quickened, and it subsides as the action of the organ becomes tranquil. M. Bouillaud is of opinion that the reduplication of the first sound is caused by the ventricle contracting twice in succession, in consequence of its inability to expel its contents; and that the reduplication of the second sound may be explained in the same way. Others have accounted for it by supposing a want of synchronism between the movements of the right and left ventricles. From the manner in which the muscular fibres of the two ventricles are arranged, it is difficult to understand how this could occur in the healthy heart; but if the right ventricle is much hypertrophied, and dilated, while the left remains of the normal size, such an occurrence is possible. In two cases recently under my care, where the triple sound was very well marked, (the reduplication being in the first sound), and where the opportunity was afforded for a post-mortem examination, I found the right ventricle much hypertrophied and dilated; in each forming the apex of the heart; the left ventricle was normal in one, and slightly dilated in the other, but the valves at the left side of the heart were sound, and no murmur had been audible in either case.

An additional sound is sometimes produced by the apex of the heart impinging against the inferior edge of the fifth left rib during the ventricular systole, which is not to be confounded with the phenomenon just described.

#### ABNORMAL SOUNDS DEVELOPED DURING THE HEART'S ACTION.

The sounds which either replace or accompany the normal sounds of the heart, and which are new, morbid, or abnormal sounds, were first named *murmurs* by Dr. Forbes, and this term has been very generally adopted since. They present different characters in different cases of disease, being sometimes blowing, sometimes resembling rather the sounds of sawing, filing, grating, whistling, &c.: and this circumstance has been deemed by some writers of sufficient importance to form the ground of their classification, and they have been divided into the "sounds of blowing," and "sounds of friction;" but, as in every instance, they are the result of *friction*, generally between a liquid and a solid, sometime between two solids, they would be more correctly classified ac



cording to their seat. Thus, sometimes they have their seat exterior to the heart, and between the opposed serous surfaces of the pericardium: these are the pericardial friction-sounds, or the peripheral or exocardial murmurs. Sometimes they have their seat within the heart, and at the orifices of the ventricles: these are the endocardial, or valvular murmurs. Sometimes, again, they have their seat in the large arteries; at others, in certain veins: the former are the arterial, the latter the venous murmurs.

These abnormal sounds will be considered here in the following order:

1. The pericardial or exocardial murmurs, or the peripheral friction-sounds; which accompany the movements of the heart, but do not interfere with its intrinsic sounds.
2. The endocardial or valvular murmurs, subdivided into the systolic and diastolic, which either take the place of the normal sounds of the heart, or prevent them from being heard.
3. The arterial murmurs.
4. The venous murmurs.

I shall now describe these sounds; to be appreciated, however, they must be heard; no verbal description can supply its place.

#### PERICARDIAL FRICTION-SOUNDS.

The abnormal sounds which have their seat between the opposed serous surfaces of the pericardium are termed attrition murmurs, or friction-sounds, from their character; and pericardial, exocardial, or peripheral murmurs, from their seat. Although all abnormal sounds are the result of friction, the exocardial are best entitled to the name, because the friction here takes place between two solids; in the others, one of the agents in the production of the sound is a liquid.

In a state of health, the opposed serous surfaces of the pericardium being exceedingly smooth, and constantly moist, glide over one another, during the motions of the heart, without producing any sound, or, at least, any which can be detected by the ear applied to the parietes of the chest. When, however, as the result of inflammation, lymph is effused upon this membrane, and its opposed surfaces become rough, unequal, or irregular, friction to a greater or less degree takes place during the movements of the heart; and this friction developes sound, which becomes

audible when the stethoscope is laid upon the parietes, and is frequently sufficiently strong to communicate a distinct vibratory sensation to the hand, as first pointed out by Dr. Stokes; and according as the action of the heart is violent or feeble, and as “the lymph effused is small in quantity, partially deposited, and forms a thin, smooth layer, or is copious, hard, and irregular, the sounds will vary from the slightest degree of rubbing, to a loud, harsh, grating, or creaking sound.”

The attention of the profession was first called to the friction-sounds of pericarditis by Dr. Stokes. M. Collin,† it is true, had described the bruit de cuir neuf, one of the rarest of these sounds, and Broussais‡ had noticed a friction-sound in pericarditis similar to that of two dry bodies, like parchment, rubbing against one another; but their remarks attracted no attention: it was reserved for Dr. Stokes to point out the distinctive auscultatory signs of the two forms of pericarditis; to determine those which belong to the variety of the disease in which the opposed surfaces of the pericardium are coated with lymph; and to demonstrate that this form of pericarditis, which heretofore had been supposed to yield no stethoscopic signs, was really the one in which auscultation is of the greatest assistance to the diagnosis.

The friction-sounds of pericarditis have received various names. They may all, however, be included under the following heads, originally laid down by Dr. Stokes:

1. “*A slight friction-sound*, perceptible only at the very commencement and at the termination of each diastole and systole of the heart.”

2. “*A rasping-sound*, very similar indeed to that produced in the worst cases of ossification of the valves. In others the sound is similar to the frottement of pleurisy, only modified by the action of the heart.”

3. “The sound resembling *the creaking of new leather*.”

*The slight friction-sound*, the “bruit de frôlement” of the French, resembles the sound produced by rubbing the hands together, or the rustling noise produced by crumpling the paper of which bank-notes are made (to which M. Bouillaud compares it),

\* Dublin Journal of Med. Science, September, 1833.

† Les Diverses Méthodes d'Exploration de la Poitrine. Paris 1824.

‡ Commentaires des Propositions de Pathologie, tome i. 1829.

or the rustling of silk ; or, when stronger, the crackling of parchment. According to Dr. Hope, this sound is produced by the presence of soft wet lymph upon the opposed surfaces of the pericardium. It appears probable, that when this sound is alone heard, and where all through it preserves the same character, it is due to the lymph being deposited merely upon one surface of the pericardium.

*The rasping sound.*—The *bruit de râpement* or *bruit de frottement* of the French, has, as its name denotes, a harsh rasping or grating character ; it often resembles accurately the rasping or the sawing of wood. The sound which it appeared to me most closely to resemble on several occasions was that which would be produced by scratching with the nail the surface of dry bone. It constitutes the to-and-fro rubbing sound of Dr. Watson. Dr. Hope refers this sound to the presence of firm and rugged lymph, the sound being loud in proportion as the lymph is rough, and the action of the heart strong.

*The creaking sound of new leather*, the *bruit de cuir neuf*, is one of the rarest of these sounds ; it resembles, accurately, the creaking of a new saddle, but is not so loud ; it may be imitated, Dr. Hope observes, “by rubbing together the fingers made sticky by resin.” I had lately a case in hospital where a sound very similar to this was developed in the bronchial tubes, owing to their obstruction by tough mucus : here, however, it of course accompanied only the respiratory movements. This creaking sound was supposed by Dr. Hope to depend upon the adhesive nature of the lymph, and to be an indication that adhesions were about to take place. More recent observations have, however, shown that this is not necessarily the case. Dr. Copland\* refers it to thickening or condensation of the sub-serous and serous tissues of the pericardium, especially of that portion reflected over the heart ; and the formation of a dense and elastic false membrane.

Another sound has been described by Dr. Hope under the name of the “continuous rumbling murmur,” which he refers to the presence of a small quantity of fluid in the sac of the pericardium, the opposed surfaces of which are also coated with lymph. He supposed that the agitation or “churning of the fluid during the motions of the heart produces the sound.” It is more rare

\* Dict. of Pract. Medicine, vol. ii.

than any of the other pericardial murmurs, with the exception perhaps of the *bruit de cuir neuf*. Finally, Dr. Walshe has recently described, in addition a “clicking murmur,” and a “murmur produced by bending of layers of exudation matter.”

*Conclusions respecting the Pericardial friction-sounds.*

When I come to speak of pericarditis, I shall have occasion again to recur to these sounds. It will be sufficient here to observe—

1. That they are almost limited to cases of inflammation of the pericardium ; that they are pathognomonic of one form, and constitute the most valuable diagnostic signs of it.

2. That they have more or less, a friction or attrition character.

3. That they are generally double, and are sometimes louder during the diastole than the systole of the ventricles.

4. That they appear to be superficial and near, and are seldom audible beyond the limits of the præcordial region.

5. That they do not, like the murmurs next to be described, replace the ordinary sounds of the heart, but are independent of them.

6. That their duration is usually short ; frequently ceasing entirely after having been heard for a few days, and not unfrequently changing their character or seat within the period that they are audible.

7. That they are frequently accompanied by a peculiar vibratory thrill sensible to the hand laid upon the parietes.

8. That a *bruit de soufflet* at one of the orifices of the left side of the heart (the result of endocarditis), not unfrequently coincides with these sounds.

ENDOCARDIAL OR VALVULAR MURMURS.

The abnormal sounds which have their seat at the orifices of the ventricles, and which replace or accompany the normal sounds of the heart, constitute the most valuable signs of disease of the valves or orifices of the left side of the organ. They present several varieties—being sometimes blowing, when they constitute the varieties of *bruit de soufflet* ; sometimes rough and harsh, resembling the familiar sounds of sawing, rasping, or filing ; and at other times having a whistling or musical character, constituting varieties of the musical murmur.

*Bruit de soufflet* or bellows-murmur, (as the name denotes,) has a blowing character, and is by many degrees the most frequent abnormal sound heard in diseased states of the valves or orifices of the heart. It was discovered and named so, by Laennec, from its similarity to this well-known sound, to which it often bears a most ridiculous resemblance. *Bruit de soufflet* does not always however, present the same character: sometimes it is perfectly smooth and soft; at other times it is loud and rough; sometimes it is a mere whiff: at others it is prolonged and lengthened out. It may accompany or replace either the first or the second sound of the heart, or it may pass into a sawing, rasping, or musical murmur. In the vast majority of cases of disease, it is limited to the left side of the heart.

*Circumstances under which Bruit de Soufflet is heard.*—*Bruit de soufflet* is heard in various and different lesions of the valves and orifices of the heart; it is not, however, limited to diseased states of this organ, but becomes audible under a variety of other circumstances. Thus, it is heard when there is any obstacle to the free passage of the blood through the orifices of the left side of the heart, or when the valves imperfectly perform their functions, and permit regurgitation. It is heard in aneurism of the left ventricle and in congenital malformations of the heart, where a communication exists between the ventricles. It is heard in cases where the lining membrane of the arch of the aorta is diseased, and in aneurism of the large arteries. It is heard in aneurismal varix, where a communication exists between an artery and a vein; in varicose aneurism where a small sac exists between them, and in the pulsating form of aneurism by anastomosis. It is frequently heard in cases where no disease of any kind exists in the heart or arterial system, but where the quality of the blood is altered; where this fluid has become more watery and less viscid than natural. Finally, it can be produced at any time in the heart and arteries by suddenly abstracting a large quantity of blood, as first pointed out by Dr. Marshall Hall; or in a large artery by making pressure upon it so as to diminish its calibre.

*Bruit de soufflet* will be heard when a tumor of any kind comes to press upon the aorta or its branches, or upon the pulmonary artery; or in malformation or deformity of the chest, from disease of its bony parietes, by which the heart's movements are impeded

or the large vessels are compressed. *Bruit de soufflet* is said to be sometimes heard for the first time, a short period previous to death, in pulmonary or cardiac disease: M. Bouillaud refers it then to the formation of fibrinous concretions in the cavities of the heart, which interfere with the action of the valves, or obstruct the orifices. It is temporarily heard in violent palpitation in hysterical subjects, or in attacks of palpitation in cases when the left ventricle is hypertrophied, and its cavity dilated: here it disappears as the palpitation subsides. *Bruit de soufflet*, in a very marked form, is likewise heard from the fourth month to the end of pregnancy: here its site is usually the iliac fossa, sometimes on both sides, more frequently only on one side.

Dr. Graves\* has noticed the remarkable fact, that a *bruit de soufflet* is sometimes audible, in the second stage of pneumonia over the affected lung, which disappears as the symptoms of the inflammation subside: it is not perceptible in the subclavian or carotid arteries of the same side, and probably, when present depends upon pressure on the branches of the pulmonary artery. Dr. Latham† has called attention to the circumstance that gentle *bruit de soufflet*, which coincides with the ventricular systole, is heard occasionally in phthisis: it is not perceived in the præcordial region, but in a circumscribed space above it on the left side. M. Zehetmayer‡ has confirmed the correctness of Dr. Latham's observation, and would appear to have noticed the murmur previous to the publication of Dr. Latham's work. He says, "I have repeatedly heard, in phthisical persons, in the second intercostal space, a decided bellows-murmur, instead of the first sound of the pulmonary artery; and in a place where there was no doubt that tubercular infiltration was present. No alteration in the vessel could be detected on post-mortem examination. Dr. Latham supposes that this murmur may have its cause in the pressure exercised by the diseased lung upon the pulmonary artery, and its first branches. In both the latter cases, the condensed lung, having become a better conductor of sound, conveyed a murmur to the ear which would probably be inaudible if the lungs were healthy. M. Gendrin§ has called attention to the circumstance that, in the cold stage of intermittent fever, a *bruit*

\* Clinical Lectures, vol. ii.      † Lectures on Diseases of the Heart, vol. i.

‡ Die Herzkrankheiten, 1844.      § Leçons sur les Maladies du Cœur, tome i.

de soufflet, analogous to that heard in cases of chlorosis, is audible on auscultation of the heart. As individuals who have been the subject of ague for any length of time are, however, in general more or less anæmic, the murmur is probably due to this cause.

*Conditions under which Bruit de Soufflet is developed.*—

Whenever bruit de soufflet is heard in the heart, it arises either from contraction or some other diseased state of the valves or orifices of the heart, which impedes the free passage of the blood; from a condition of the valves which prevents them closing the orifice, and permits regurgitation; from the blood being impelled with increased force and velocity through the aortic orifice, or through an abnormal orifice, as in aneurism of the left ventricle, and congenital malformations of the heart; from some alteration in the quality of the blood itself, or in its quantity; from the formation of fibrinous or other concretions in the cavities of the heart, which interrupt the play of the valves, or impede the orifices; or where morbid growths are developed in the cavity of the chest, which compress or displace the heart; or where the bony walls of the thorax are deformed, and this cavity is much narrowed.

Whenever bruit de soufflet is audible in the arteries, it arises either from roughness of the lining membrane of the vessel; or from the calibre of the artery being diminished, owing to pressure upon the part; or from an altered condition of the blood; or from an aneurismal sac springing from a large artery; or where an abnormal communication exists between a large artery and a vein; or where an aneurismal sac is seated between an artery and a vein, through which the blood passes from the one to the other.

Bruit de soufflet is therefore a symptom in endocarditis, where the aortic or mitral valves are rigid, thickened, or indurated; or where vegetations form upon them, or upon the orifices; as well as in the chronic forms of disease of the valves or orifices, accompanied by contraction of the orifices, by ossific or other deposit upon the valves, or adhesion of the valves to one another or to the parietes. It will likewise be heard where rupture of a valve or of a tendinous cord takes place, or in cases of congenital malformation of the valves, as where a cribriform condition of their curtains exists. Bruit de soufflet is likewise a symptom in dilatation of the orifices of the left side of the heart without any disease of the valves, or where they are prevented by any other cause



from perfectly fulfilling their functions. It is a symptom in aneurism of the left ventricle ; in congenital malformations of the heart ; in cases of morbid growths in the cavity of the thorax ; in aneurism of all the large arteries ; in aneurismal varix, and varicose aneurism ; in disease of the lining membrane of the arch of the aorta and, in anæmic states of the system.

*Mechanism of production of Bruit de soufflet.*—Laennec attributed bruit de soufflet to spasm, or to a vibratory action of the heart ; Bertin, to the increased friction produced by the passage of the blood through a contracted orifice. Other physiologists have endeavoured to explain it by referring it to increased velocity of the circulation, or to very energetic action of the heart, or “a certain resistance given to the blood moving with a certain force.” Dr. Leared\* regards bruit de soufflet as “the result of diminution mutually exerted in the pressure of the particles of body of fluid in motion, the consequence of which, is, the main current becomes split into numerous smaller ones, the particles place of a progressive motion, in which an equable relation each other was preserved, assume new and irregular movements. Others have referred it to modifications or alterations in the tissue or structure of the parietes of the heart, or to morbid conditions of the blood itself. M. Bouillaud† is of opinion that bruit de soufflet may be produced under three conditions—viz. increased action of the heart ; narrowing of any portion of the canal through which the blood passes ; or roughness of the surface over which the blood flows. Dr. Corrigan,‡ who has written at length upon this subject and who made several experiments with the object of determining the question, arrived at the following conclusions respecting the conditions necessary for the production of this sound.

“1. A current-like motion of the blood (instead of its natural equable movement), tending to produce corresponding vibrations of the sides of the cavities or arteries through which it is moving ;” and

“2. A diminished tension of the parietes of the arteries or cavities themselves, in consequence of which their parietes are easily thrown into vibrations by the irregular current of the contained fluid ; which vibrations cause, on the sense of touch, frémissement—and, on the sense of hearing, bruit de soufflet.”

\* Dublin Quarterly Journal, May, 1852. † Archives Gén. de Médecine, tome xi

‡ Dublin Journal of Medicine, vols. x. and xiv.



In every instance in which bruit de soufflet is audible, it is clear that some physical agent, capable of producing it, must be in operation : now the only physical agent that is invariably in operation, and under every variety of circumstances where bruit de soufflet is heard, is increased friction between the blood and the parts along or through which it passes ; which appears to be amply sufficient, not only to develop a murmur, but to convert the normal sounds of the heart into one. Thus :

1. When the orifice of the aorta is contracted, or its valves are diseased, so as to impede the direct passage of the blood, there must necessarily be increase of friction ; and the normal first sound of the heart will be converted into a murmur.

2. When the mitral valve is diseased, and permits a reflux current of blood into the left auricle at each ventricular systole, a murmur will be developed, which, from its louder tone, will obscure or render inaudible the first sound of the heart.

3. When the semilunar valves of the aorta imperfectly close the orifice, the blood must regurgitate from the aorta into the left ventricle at each ventricular diastole ; and that this occurs with sufficient force to generate a murmur, which obscures or renders inaudible the normal second sound of the heart, is well known.

4. When the left auriculo-ventricular orifice becomes dilated, and the valve cannot, from this cause, fulfil its function perfectly, regurgitation of the blood occurs at each ventricular systole, and a bruit de soufflet is developed, which is usually sufficiently strong to obscure the normal first sound of the heart.

5. When the blood is propelled with greatly augmented force, and with increased velocity, through the aortic orifice, the amount of friction between the blood and the parietes of this orifice must be increased ; and if it is sufficiently strong to generate a murmur, this will have the character of bruit de soufflet, and will take the place of the normal first sound of the heart.

6. When the lining membrane of the ascending portion of the arch of the aorta loses its natural smoothness and polish, the friction between the blood and the part along which it passes must be increased ; and, if the heart's action is strong, a murmur will be developed, which will be synchronous with the first sound of the heart, and will often be audible in the large vessels which come off from the arch of the aorta.

7. When the viscosity of the blood is diminished, and its watery parts are increased, a greater amount of friction will take place between this fluid and the orifice of the aorta, in its passage out of the ventricle, as well as in the large arteries, which is usually quite sufficient to convert the normal first sound of the heart into a murmur.

8. When the blood is propelled into an abnormal cavity, or through a preternatural orifice, and the action of the left ventricle is strong, the friction will be sufficient to generate a murmur which will obscure the first sound of the heart.

9. When the large arteries in the cavity of the thorax are compressed by the growth of a tumor within, or by deformity of the bony cavities from disease without, the diminution of their calibre is often sufficient to generate a murmur, which will have the character of bruit de soufflet.

10. When fibrinous concretions form in the cavities of the heart and obstruct the orifices, or impede the movements of the valves, if the action of the heart is strong, sufficient friction may be produced to develop a murmur; but as this scarcely ever occurs, except during the last few hours or days of life, the current of blood is generally too feeble to develop a murmur.

Lastly, the bruit de soufflet, heard after the fourth month of pregnancy, when the uterus has risen out of the pelvis, and which is generally, but incorrectly, termed the *placental souffle*, has its cause partly in an altered condition of the blood, but principally in the pressure exercised by the enlarged uterus upon the iliac arteries, or their branches; indeed Professor Von Kiwisch places its seat, exclusively, in the epigastric arteries. Thus it always accompanies the ventricular systole, it is audible usually only in one iliac fossa: and an exactly similar murmur has been heard by Dr. Montgomery\* in cases where the uterus itself was enlarged from disease, or where an abdominal tumor compressed the aorta. In addition, M. Bouillaud has found that, by changing the position of the patient, the site of the murmur is altered; and Dr. Cowan† observes that the bruit de soufflet, in these cases, "can be detected in the femoral arteries immediately below the arch," and that "the sound may, by change of position, be transferred from one femoral artery to the other, always corresponding with the side of the uterine murmur."

\* Cyclopæd. of Pract. Med., art. Pregnancy. † Medical Gazette, vol. xvii. 1836.

In the majority of the foregoing examples the bruit de soufflet is heard at the period of the ventricular systole, and replaces or accompanies the first sound of the heart; the force with which the blood is propelled by the left ventricle being much greater than that with which it enters it, the friction between the blood and the parts along or through which it passes must be much more considerable in the former than the latter. In the former it is generally sufficient to generate a murmur, in the latter it only occasionally does so: and, when a murmur is developed, it has a different character. For instance, when the mitral valve or orifice is diseased, so as to permit regurgitation, a bruit de soufflet will be heard at the period of the ventricular systole, which quite obscures the normal first sound of the heart; when the aortic valves permit regurgitation, a bruit de soufflet is audible at the period of the ventricular diastole, which likewise obscures the normal second sound of the heart. But the two murmurs have a different character, that which accompanies mitral regurgitation is usually loud, strong and blowing; that which accompanies aortic regurgitation is usually soft and whispering, because the force with which the blood is propelled from the ventricle is much greater than that with which it enters it.

Two or more of the conditions described above are not unfrequently combined in the same subject; and we may have a murmur taking the place of, or obscuring both sounds of the heart; or the bruit de soufflet may pass into a sawing, rasping, or musical murmur; or, finally, the valvular disease may be complicated with an anæmic state of the system, by which the difficulty of the diagnosis is sometimes considerably increased.

*Bruit de Soufflet at the right side of the heart.*—The foregoing remarks have reference, more particularly, to bruit de soufflet at the left side of the heart: in fact, this abnormal sound is very rare at the right side, because the force with which the blood enters or is expelled from the right ventricle is generally too feeble to generate a murmur; while valvular disease at this side of the heart is uncommon. Dr. Clendinning\* has shown, from statistics in 100 cases, that valvular disease is sixteen times more frequent at the left than at the right side of the heart. When we consider, too, that in cases of anæmia the murmur is limited to the left side,

\* Medical Gazette.

and that murmurs from regurgitation are scarcely ever produced at the right side of the heart, we can understand why *bruit de soufflet* should be so rare at this side. For instance mitral regurgitation is one of the most frequent causes of *bruit de soufflet* at the left side of the heart: on the other hand, tricuspid regurgitation is even more frequent; but it is not accompanied by any morbid sound, and is usually recognised by another sign—viz. jugular pulsation. Again, aortic regurgitation is a frequent cause of a murmur at the left side of the heart, but a very uncommon cause of a murmur at the pulmonary orifice. “I believe (Dr. Hope\* observes) diastolic murmur of the pulmonic valves to be exceedingly rare from disease of the valves themselves, as I have never met with a case, or been able to find one recorded.” “From a rude numerical calculation (he adds) deduced from the cases that I have seen, I should think that there would be at least thirty chances to one against a murmur connected with the semilunar valves being seated in the pulmonic set.”

Although the second sound at the right side of the heart is scarcely every converted into a murmur, owing to regurgitation through the sigmoid valves of the pulmonary artery, this sound is sometimes altered, augmented, or rendered sharper, owing to hypertrophy with dilatation of the right ventricle, consequent upon disease of the mitral valve, as first pointed out by Skoda and Zehetmayer.† According to Skoda, when the left auriculo-ventricular valve permits regurgitation, the second sound at the pulmonary orifice becomes more intense, or sharper; which he explains by the impeded circulation through the lungs reacting upon the right side of the heart, in consequence of which the blood in the distended pulmonary artery reacts with unusual force upon the sigmoid valves of this vessel. He is even of opinion that, from this augmentation of the second sound at the pulmonary orifice we might diagnose disease of the mitral valve or orifice previous to any abnormal sound being heard at it. Zehetmayer considers that this phenomenon is never absent in cases of mitral regurgitation and that the diagnosis of the latter cannot be positively made unless it is likewise perceived.

A murmur at the pulmonary orifice, or in the pulmonary artery, accompanying the ventricular systole and the first sound

\* Treatise on Diseases of the Heart, 4th edit. † Die Herzkrankheiten, 184

is less rare than any murmur at the right side of the heart. It seldom, however, depends upon disease of the sigmoid valves; in general it arises from the presence of a tumor, or from a diseased lung compressing the pulmonary artery. Dr. Hope relates a case in which an aneurism at the origin of the aorta compressed the pulmonary orifice, and contributed to produce a systolic murmur; and another, where extensive ossification of the pulmonary artery, gave rise likewise to one. Dr. Elliotson met with two cases where portions of cartilage in the pericardium pressed upon and contracted the pulmonary artery so as to produce a murmur.

The value of bruit de soufflet as a sign of disease of the valves or orifices of the heart, depends,

1. Upon the period of the heart's action at which it occurs.
2. Upon the situation at which it is best marked.
3. Upon its character or tone.
4. Upon its being constantly present or not.

In organic disease of the valves and orifices of the heart, bruit de soufflet is in a great measure limited to the left side of the organ and has its seat either at the aortic or mitral orifice. When it replaces the first sound of the heart, it depends, in almost all cases, either upon obstructive disease of the aortic orifice, or regurgitant disease of the mitral orifice. When it occurs at the period of the ventricular diastole, and of the second sound of the heart, it almost always has its cause in a state of the aortic valves permitting regurgitation. When bruit de soufflet accompanies both the systole and the diastole of the ventricles, it may have its cause either in obstructive disease of the aortic orifice, combined with a state of its valves permitting regurgitation; or in the mitral and aortic valves, both permitting regurgitation.

The first sound of the heart is not unfrequently replaced by bruit de soufflet when no disease of the valves or orifices exists; but a murmur is never heard at the period of the second sound of the heart, unless in cases of disease. A murmur at the right side of the heart is, as has already been observed, extremely rare; and when it is heard, it will be found to depend more frequently upon disease of other parts, than of the valves or orifices of the right ventricle.

## SAWING, FILING, AND GRATING VALVULAR MURMURS.

The valvular or endocardial murmurs next to be considered have a harsh, rough, or grating character : they were first described by Laennec and have received various names : as the “sawing murmur” (*bruit de scie*) ; the “rasping or grating murmur” (*bruit de râpe*) and the “filing murmur” (*bruit de lime*) ; which more or less resemble these familiar sounds. Although the rough valvular murmurs are distinguished by different names, they are by no means to be regarded as characteristic of distinct forms of disease ; in fact, they are nothing more than degrees or varieties of rough sounds, having the same origin as *bruit de soufflet*, although less common than it, and more decidedly pathognomonic of organic disease of the valves or orifices of the heart.

It is a common opinion, that the harsh, grating, valvular murmurs are the result of cartilaginous or calcareous degeneration of the valve at which they have their seat, and that they always indicate a more advanced state of disease of the valves, or a greater amount of contraction of the orifice than *bruit de soufflet*. This was the opinion of Laennec, and this appears to be the view taken by M. Bouillaud ; it is far, however, from being correct ; these sounds are heard in cases where the valves have undergone neither calcareous or cartilaginous degeneration ; and a very advanced state of disease of a valve is often indicated simply by *bruit de soufflet*, while in a still more advanced stage it may disappear, and no murmur of any kind be audible. This is sometimes observed at the mitral orifice, when the contraction becomes so extreme as not to permit a sufficient current of reflux blood through it to develop a murmur.

Although these harsh grating sounds do not necessarily indicate either calcareous or cartilaginous degeneration of the valves, they are often not heard in cases where valvular disease is much advanced ; yet, when they are well marked, we may conclude that the degree of friction between the blood and the parietes of the orifice at which they are developed, is greater than when simply *bruit de soufflet* is heard. In the majority of cases this is owing to hypertrophy, or hypertrophy with some dilatation of the ventricle, complicating the valvular disease, by which the contracting power of the ventricle is increased, and the blood is transmit-

with augmented force through the diseased orifice. Hence the rough valvular murmurs are loudest at the period of the ventricular systole and first sound of the heart, and are not heard in inorganic affections of the organ, in which bruit de soufflet is so common.

In almost every instance, too, they are preceded by bruit de soufflet; the one insensibly passes into the other; and they are to be regarded as merely more intense degrees of the latter.

*Conclusions from the presence of a Rough Valvular Murmur.*

1. The harsh, grating valvular murmurs are to be regarded as nothing more than bruit de soufflet exaggerated.

2. They are limited to the left side of the heart, and are heard only in cases of organic disease of the valves or orifices.

3. They are often audible only at the period of the ventricular systole, and they then generally indicate obstructive disease of the aortic orifice.

4. When heard at the period of the second sound, they are always accompanied by a rough systolic murmur; and they have their cause, then, in a combination of disease of the coats of the aorta, with regurgitant disease of the semilunar valves.

5. In almost every instance, hypertrophy with dilatation of the left ventricle, accompanies the valvular disease.

6. They do not necessarily indicate osseous or calcareous degeneration of the valves.

MUSICAL VALVULAR MURMUR.

The musical murmur of the heart—the “bruit de soufflet musical ou sibilant” of the French writers—is a very remarkable and rather rare sound: it resembles sometimes, as the name denotes a whistle: sometimes the notes of a wind instrument, or the cooing of a dove. It may consist of only a single note, or of two or three; seldom more: it is sometimes so loud as to be audible without the stethoscope, and at some distance from the patient; and it may accompany either the systole or diastole of the ventricle.

We are indebted to Dr. Elliotson\* for the earliest description

\* Lumleyan Lectures.



of this murmur : he first called attention to it as a sign of valvular disease, and described the particular lesion of the valve in which he had met with it. Laennec says he never heard a musical murmur in the heart, but he has described a musical murmur in the arteries : the latter, however, we know now has its seat in the veins.

The musical valvular murmur is to be regarded as not more than a variety of bruit de soufflet ; which precedes ; and usually takes its place when it intermits or subsides, or when the exciting cause has been removed. In fact, as both Dr. Hope and M. Bouillaud remark, “there is no greater difference between two sounds than there is between blowing with the lip and whistling.” Thus, in a case which was in hospital some time since, a loud musical murmur was heard upon the day of the patient's admission, after the exertion of walking to the hospital ; on the day following, when the circulation had become tranquil, this murmur had disappeared, and was replaced by bruit de soufflet. On a post-mortem examination, the valvular lesion was found to consist in a cribriform condition of the curtains of the mitral valve by which regurgitation had been permitted. In a case of acute endocarditis, which was in hospital a short time since, bruit de soufflet was first heard ; and when the patient had been subjected to treatment for a short time, this was replaced by a musical murmur, which continued to be audible as long as the patient remained under observation.

Like the sawing, and other rough valvular murmurs, the musical murmur is almost always a sign of organic lesion of the valves or orifices of the left side of the heart ; and, so far as we have had the opportunity of observing, it is limited to the regurgitant lesions of the aortic and mitral valves. This murmur is frequently accompanied by fremitus catenatus, and is not audible in any case in which the semilunar or the mitral valve imperfectly fulfil their functions, provided the aperture through which regurgitation occurs is *small*, and that the heart beats vigorously.

As regurgitation cannot occur in a healthy condition of the valves, and, as the musical murmur is in a great measure confined to cases of regurgitation, it is a valuable physical sign of valvular disease, when present, and it may be regarded as almost pathognomonic.



nomonic, if combined with other symptoms of organic disease of the heart.

The division of abnormal sounds, which we have next to consider, have their seat in the arteries, or in the veins, or in both. They all, with one exception, come under the head "*inorganic murmurs*." They are,

1. The arterial bruit de soufflet.
2. The rough, grating, arterial murmur.
3. The continuous venous murmur, or "bruit de diable."
4. The musical venous murmur.

#### ARTERIAL MURMURS.

The abnormal sounds which have their seat in the arteries may, like those developed at the orifices of the heart, have either a blowing or a rough grating character. It is only in the large arteries that they are heard; and in investigating them it is always necessary to employ the stethoscope, indeed, M. Vernois has proposed a modification of this instrument for the sole purpose of examining the arteries, which consists in "excavating the two opposite points of its circumference for the reception of the vessel subjected to examination," by which the pressure upon the artery, caused by the stethoscopes ordinarily employed will be obviated. This appears to be an unnecessary refinement, as the instruments in common use are quite sufficient for every purpose.

*Arterial Bruit de Soufflet*.—The abnormal sound heard most frequently in the arteries is bruit de soufflet. In a state of health, the blood, in its passage through the large arteries, causes such slight friction that very little sound is produced: this is short, single, and repeated at equal intervals, from sixty to eighty times in a minute, and is synchronous with the ventricular systole and with the pulse. If slight pressure be made upon the vessel with the finger, or with the stethoscope, this sound increases in intensity; and, if the pressure be increased, it is converted into a murmur having the character of bruit de soufflet. Increase of friction between the blood and the lining membrane of a healthy vessel, produced by merely diminishing the channel through which the current flows, is therefore sufficient to develop a murmur in a large artery.

Again, one of the most important properties of the blood is its

viscosity. In some morbid conditions of the system this property is impaired; the blood becomes impoverished, attenuated, and watery, the red globules diminish, serum superabounds, and, as a natural consequence, the viscosity of this fluid is less than natural. In other cases, the actual amount of blood in the system is diminished, as after profuse hæmorrhage, or when venæsection has been carried to an extreme degree. In both cases, the amount of friction between the blood and the lining membrane of the arteries must necessarily be greater than natural; and this increased friction is capable of developing a murmur analogous to that produced by pressure upon a healthy vessel. In order that the murmur be well marked, it is necessary that a certain degree of velocity of the current above the natural standard should occur in addition.

The arteries in which *bruit de soufflet* is most commonly heard are the aorta, the carotids and subclavians, the iliac and femoral arteries. It may be audible in any of these vessels, when the blood is propelled with increased force and velocity through them; when the calibre of the artery is diminished by pressure from without; when the coats of the arteries have lost their natural and healthy state of tension; or when the blood is more or less attenuated, owing to its watery parts being increased, and the red globules diminished, the result either of profuse hæmorrhage, or of a chlorotic state of the system. The latter are frequently accompanied by the abnormal sounds (presently to be described) which have their seat in the veins, and the arterial *bruit de soufflet* is sometimes accompanied by a murmur at the aortic orifice, depending upon the same cause.

The *bruit de soufflet*, which has its seat in the large arteries, has been mistaken for a valvular murmur, particularly when heard over the arch of the aorta, or in the carotid and subclavian arteries.

It may be distinguished,

1. By its character, which, as Dr. Hope observes, is “usually a mere whiff.”

2. By not being heard at the period of the ventricular diastole and the second sound of the heart.

3. By being audible in several arteries at the same time.

4. By the facility with which the murmur is altered by varying the pressure of the stethoscope, by which it may be converted into a hissing or whistling murmur.

5. By not being constantly present: by subsiding occasionally when the circulation becomes tranquil, and returning when palpitation ensues.

6. By the general signs of anæmia being present.

7. By the absence of the physical or general signs of organic disease of the heart.

8. By entirely disappearing under treatment calculated to relieve the anæmic state of the system.

*Rough or grating Arterial Murmurs.*—It is a very general opinion, that bruit de soufflet is the only abnormal murmur developed in the large arteries. This, however, is far from being the case: a rough, sawing murmur is not unfrequent; but its seat is limited to the arch of the aorta, and it is not confined to the period of the ventricular systole, but it frequently accompanies the ventricular diastole likewise. It is probably owing to these circumstances that the phenomenon has been overlooked, the murmur, when heard, having been confounded with a cardiac murmur.

In a healthy state of the arterial system, the arteries, we know, are constantly filled; when the amount of blood transmitted to them is increased, the artery dilates, in order to accommodate itself to the increased quantity; when the amount of blood is diminished, the arterial coats contract upon their contents, and the calibre of the artery is diminished in proportion; but, whether the amount of blood is greater or less, these vessels are always full. This depends upon the peculiar property possessed by arterial tissue, usually termed elasticity, but which is evidently something more than mere elasticity, which is a property common to dead as well as living tissues. The aorta, in a healthy state, possesses this peculiar vital property in a high degree; which is due, not only to its proper fibrous coat, but to its subserous or *sclerous* coat, which, in this artery is very well developed, as first described by Dr. Chevers.\*

It not unfrequently happens that the coats of the arch of the aorta suffer from disease, which impairs its elasticity; the tube becomes rigid, and its calibre cannot alter with the increased or diminished amount of blood transmitted to it. At the same time, the natural smoothness and polish, which its lining membrane presents in the healthy state, is diminished; and, the interior of the

\* Guy's Hospital Reports.

artery becomes rough and uneven from adventitious deposit; while very generally its calibre becomes increased.

During the systole of the left ventricle, the arch of the aorta is necessarily more distended than during the ventricular diastole: in the morbid condition under consideration, however, as its coats are incapable of contracting upon their contents, and as the calibre of this part of the vessel remains the same at these different periods of the heart's action, a vacuum would occur here when the ventricular systole ceases, did not the blood regurgitate from the carotic and subclavian arteries to occupy it; and this can scarcely occur without producing sound, which will, of course, accompany the ventricular diastole and the second sound of the heart.

In this diseased state of the arch of the aorta, not only is the artery rigid and inelastic, but often its interior, owing to adventitious deposit, becomes uneven and rough; there will therefore necessarily be increased friction between the blood and the lining membrane of the vessel, and a murmur will be developed, which will be heard at the period of the ventricular systole, and will be synchronous with the first sound of the heart, and with the pulse. As the ventricular systole ceases, the blood from the large vessel which come off from the arch of the aorta regurgitates into it, and the passage backwards of this fluid over a similar rough surface causes a second murmur, which will, of course, be synchronous with the ventricular diastole, and the second sound of the heart.

When the stethoscope is applied over the first bone of the sternum in this diseased state of the arch of the aorta, a short double, sawing murmur will be heard, loudest over the first bone of the sternum and audible frequently as low as the base of the heart, sometimes a little lower down, but never at the apex of the organ. The first of these sounds is synchronous with the ventricular systole, and the pulse; the second, with the ventricular diastole. Both sounds appear to be near, and both are short and rough, not either prolonged or blowing. In some instances murmur is present only with the first sound, or even this may be absent, and the second sound may be neither rough or harsh. This will occur when the arch of the aorta, although rigid and inelastic, has not lost, in any great degree, its natural smoothness and polish, but still permits regurgitation into it: the double sound then heard resembles very closely the double sound of the

heart, and is commonly supposed to be it transmitted beyond its normal limits. It may even happen that the arch of the aorta is dilated, rigid, and inelastic, and its lining rough and irregular, and yet no murmur will be developed : this will occur when the heart's action is feeble from softening or other alteration of the muscular tissue of the left ventricle, or when the arterial system is congested ; or when the cavities of the left side of the heart are loaded with blood. Under similar circumstances, we know that a murmur may be absent in cases of considerable disease of the valves or orifices.

In addition to the physical signs above mentioned, this diseased state of the arch of the aorta is accompanied by the peculiar jarring of the pulse which was supposed to be characteristic of regurgitation through the aortic valves ; by visible pulsation of the carotids ; and by a visible and locomotive pulse in the arteries of the upper extremities. The peculiar jerking of the pulse, and the visible and locomotive pulsation of the arteries, depend upon the same cause as where the aortic valves permit regurgitation : here, however, the regurgitation is into the aorta itself. These signs will be more marked if the arch of the aorta, in addition to being rigid and inelastic, is at the same time dilated, and if the action of the heart is strong.

The physical signs of this diseased state of the arch of the aorta have been hitherto confounded with those of valvular disease at the aortic orifice : no writer that I am acquainted with seems to have been aware, that regurgitation into the arch of the aorta, from the larger vessels which come off from it, ever occurs, or that a backward current of blood in this part is capable of developing a murmur. This has probably arisen from the general symptoms, in the early stage, not being such as to attract particular attention ; and, as patency of the aortic valves is one of the consequences of the long continuance of this diseased state of the aorta, when the latter lesion has been found after death, the symptoms have been referred to it alone.

The form of cardiac disease with which this diseased condition of the coats of the arch of the aorta has been most frequently confounded, is a state of the aortic valves permitting regurgitation, which it resembles in a murmur synchronous with the second cardiac sound, accompanying both morbid conditions, in the jarring pulse, and the visible pulsation of the arteries of the neck

and upper extremities, which are common to both. It may, however, be distinguished by the character of the murmur with second sound, which, in this diseased state of the aorta, is sh rough, and harsh, while in patency of the aortic valves it is usu prolonged, and blowing; as well as by the situation in which murmur is transmitted to the ear. In patency of the a valves it is audible from the base to the apex of the heart; in this morbid condition it is loudest over the first bone of the s num, and it is not heard below the base of the heart; at least is very indistinct below this point. Besides, in this diseased state, when a murmur accompanies the second sound, one is likewise always heard with the first, which is not the case in the patency of the semilunar valves.

#### VENOUS MURMURS.

The abnormal sounds, which have their seat in the veins, may have either a blowing or a musical character. They all come under the class "inorganic murmurs," and have received a variety of names; as the venous murmur, or venous hum, the sour continuous blowing, bruit de diable, and musical venous murmur. They may all be included under the heads, continuous, and musical venous murmur.

*Continuous venous Murmur.*—The continuous venous murmur or venous hum, is termed so from its being a continuous uninterrupted sound, and venous from its seat. M. Bouillaud names "bruit de diable," from its resemblance to the familiar sound made in the spinning of a kind of humming top, which is known under the popular name of *diable* in France, and this name has been very generally retained since. It resembles pretty exactly the sound heard when a large univalve shell is held to the ear; it has also been compared to the noise of the sea on a distant strand, or to the buzz produced by a number of people talking together in a large room; and it may be imitated, as Hopè observes, "by forcing the breath in whispering a continuous *who*." It is often accompanied by the arterial bruit de soufflet, which appears to increase its intensity at each systole of the heart, and it often acquires increased loudness at each inspiration owing to the expansion of the thorax, which allows the blood more rapidly to descend to the right side of the heart.

The veins in which the continuous murmur is heard, are the jugulars, particularly the internal jugular, and the veins which open into them. The situation in which it is best marked is immediately above the clavicle: it is not audible close under the chin; it may often be heard at both sides of the neck, though usually it is more feeble upon one side; or it may be limited to one side, and, in my experience, it is both more frequent and more intense upon the right than the left side, though M. Bouillaud states that he most frequently finds it upon the left side. The more direct course of the veins upon the right side of the neck, to reach the vena cava, may account for its greater frequency upon the right side. The position in which to place the patient, in order to hear this murmur, is the sitting or standing posture, with the face inclined towards the opposite shoulder, and the chin somewhat elevated.

The continuous venous murmur sometimes subsides suddenly at the point where it was audible a few minutes previously; slight change of position of the patient's head will produce this effect, or will alter the character of the sound. This depends upon the pressure exercised on the vein by the parts lying over it, which varies with the motions of the head. M. Bouillaud says, that drawing the larynx towards the opposite side will diminish the murmur suddenly, or cause it to cease; it does so by putting the omo-hyoid muscle upon the stretch, by which the deep cervical fascia is rendered tense, and pressure is made upon the internal jugular vein. Altering the pressure of the stethoscope alters the character of this murmur in a remarkable manner, sometimes diminishing, sometimes considerably increasing its intensity, and rendering it hoarse, or converting it into a musical murmur: when pressure sufficient to obliterate the current through the vein is made, either with the edge of the stethoscope or with the finger above, the murmur instantly ceases.

*Musical venous Murmur.*—The musical venous murmur is less frequent than the continuous murmur: it has, however, its seat in the same vessels, is audible in the same situation and occurs in precisely the same kind of cases. It is less common in the male, and, like the continuous murmur, is more frequently heard upon the right than the left side of the neck. Like the latter, also, it intermits, changes its character, or ceases from very



trifling alterations in the position of the patient: it is always preceded by the continuous murmur, which takes its place when it subsides. In order to hear either murmur, it is essential to employ the stethoscope: and although it is sometimes audible on very gentle pressure by the stethoscope, it generally requires stronger pressure with the instrument than is sufficient to develop a continuous murmur.

The musical venous murmur sometimes resembles a whistle with the lips, or the prolonged whistling sound of a current of air through a key-hole; sometimes it is more like the chirping of a bird, or the humming of a fly. It often consists of a single note, sometimes of two, more rarely of several distinct notes. The French writers, who place its seat in the arteries, term it the chant of the arteries; and both Laennec's and Bouillaud's works contain a scale, in which they have endeavoured to represent by musical notes the melody heard in some cases.

The musical venous murmur appears to have the same relation to the continuous venous murmur as the musical valvular murmur has to the bellows murmur; the one is preceded by, and passes into the other, and the former is to be regarded as nothing more than an exaggerated variety of the latter. There is, in fact, no greater difference in the nature of the sounds, and in the mode in which they are produced, than there is (Dr. Hope observes) "between blowing with the lips, and whistling."

*Seat of the the venous Murmurs.*—The continuous and the musical venous murmurs were for a long time supposed to have their seat in the large arteries which come off from the arch of the aorta: this was the opinion of Laennec, who first discovered them; and it has been advocated by almost every French writer from him to M. Bouillaud, MM. Barth and Roger, and M. Beau, the latest authorities upon the subject. The first to correct this error was Dr. Ogier Ward;\* he it was who first demonstrated that the veins, not the arteries, were the seat of these murmurs, and he first pointed out the distinguishing marks between the venous and the arterial murmurs.

In many cases, the continuous or the musical venous murmur are accompanied by the arterial bruit de soufflet already described; they may always, however, be readily distinguished from each

\* London Medical Gazette.



other: the arterial murmur is an intermittent sound, the venous a continuous sound; the arterial murmur is a short whiffing sound, while the venous has a humming or whistling character.

That the continuous and the musical murmur have their seat in the veins is proved by their diminishing, or ceasing altogether, when pressure with the finger is made upon the vein above, or when the stethoscope is pressed firmly on it, so as to obliterate the current: the feeble bruit, which is still occasionally audible, comes from the small venous branches in the neighbourhood. M. Bouillaud, who places the seat of these murmurs in the arteries, observes that "pressure above the vessel with sufficient force to interrupt the current will cause a cessation of the murmur," which is quite true: but it is not quite so easy as M. Bouillaud seems to think, to "interrupt the current" in the carotid or subclavian arteries; the degree of pressure required would cause rather more pain than most patients would like to submit to often; while the moderate degree of pressure necessary to interrupt the venous murmur would, if it had its seat in the artery, render it louder, instead of interrupting it. In fact, the trifling pressure which is necessary to cause the cessation of these sounds is the best proof that they have their seat in the veins; and that they are not seated in the arteries, is proved by the true arterial bruit de soufflet being very often heard at the same time, and by its continuing to be audible when the venous murmur has been checked by the pressure of the finger or the stethoscope.

*Mechanism of production of the venous Murmurs.*—In previous chapters I have endeavoured to prove, that every sound, whether normal or abnormal, developed in the heart or arteries, is produced by friction between the blood and the lining membrane of the part along or through which it passes; that abnormal sounds are nothing more than exaggerated normal sounds, and that the character of the abnormal sounds will vary according to a variety of circumstances; as the roughness or smoothness of the lining membrane, the nature and amount of the obstruction to the current of blood, the rapidity and strength with which the blood is propelled, and the physical condition of the circulating fluid, particularly its density and viscosity.

In the cases in which the continuous or the musical venous murmur are heard, the organs of circulation are in a healthy state;

but the blood is altered, its density is diminished, its watery parts are increased, and its viscidility is less than that of healthy blood, while the current is more rapid than in health—conditions sufficient to develop a murmur in the arteries; and accordingly the arterial bruit de soufflet is very generally audible in such cases. The current through the veins being naturally feeble, compared to that through the arteries, their coats being lax and readily distensible, no great amount of friction can occur between the blood and the lining membrane of these vessels, even though this fluid be altered in the way that I have mentioned. In order, therefore, that a murmur should be developed in a vein, the friction between the blood and its lining membrane must be *mechanically* increased.

The murmurs which we are considering have their seat essentially in the jugular veins; principally, I believe, in the internal jugulars. Now, in order that friction sufficient to develop sound should take place between the blood and the lining membrane of these veins, it is necessary that the tissues covering them, viz., the skin, the platysma, and the cervical fascia, should be rendered tense; which is effected by turning the patient's head towards the opposite shoulder, and elevating the chin, by which a moderate degree of pressure is exercised upon these veins in their whole course in the neck, and they are placed in a favourable condition for the development of sound. If the stethoscope be now applied its pressure will slightly diminish the calibre of the vein, the friction between the blood and the lining membrane of the vessel will be increased, and a murmur will be developed, which will of course have a continuous character, because the current through the vessel is continuous. If increased pressure be made by the stethoscope, the character of the murmur will be altered; it will be rendered harsher, or hoarser, or converted into a whistle, or other musical murmur, as the case may be; while, if the stethoscope be pressed still more strongly upon the vein, so as to obliterate the current, the murmur will instantly cease.

I am therefore of opinion that the continuous and musical venous murmurs, unlike the cardiac and arterial murmurs, are not naturally present in the veins, but that they are mechanically developed by the pressure of the stethoscope, when the coats of the vein have been rendered tense, by putting the parts covering it upon the stretch. Thus, if we completely relax the fascia and

muscles of the neck, by making the patient incline the head forward, and then apply the stethoscope, the murmur will be inaudible, or scarcely heard; while, if we turn the patient's head slightly towards the opposite side, the murmur will be loudly heard, although the same degree of pressure is made by the stethoscope.

Again, the *bruit de diable* is described by writers as being often audible one day, and inaudible the next; and as disappearing suddenly at a point where immediately previous it had been loud. Now, if we examine the connections of the deep fascia of the neck with the sheath of the cervical vessels, and with the muscles, we shall perceive that a very trifling alteration in the position of the patient's head will render this fascia tense, or the contrary; and we can easily understand, therefore, how the pressure of the stethoscope may, in one position of the patient, fail to develop the murmur, while in another position, the same amount of pressure will render it loud.

The venous murmur (as has often been observed) diminishes or subsides when the face is turned quite round towards the opposite side. Now the effect of this position is to render tense both the cervical fascia and the muscles which cover the internal jugular, by which so much pressure is made upon the vein as greatly to diminish the current through it, when of course the murmur will either subside or become very feeble. The venous murmur is sometimes heard on both sides, sometimes only on one side, and is usually better marked upon the right than the left side. This is readily explained by the more direct course of the blood towards the heart on the right side. When this murmur has been only heard on the left side, the French writers (who suppose the carotid artery to be its seat) are obliged to refer it then to "some irregular distribution of the left carotid artery, by which this vessel is rendered more superficial." "We must allow (M. Beau observes) a large share to the anomalies so frequent in the arterial system."

The continuous murmur ceases or diminishes materially in the recumbent posture, and returns again in the sitting or erect posture. This may be accounted for partly by the effect of gravity upon the circulation in the jugular vein, and partly owing to the rapidity of the circulation being greater in the erect or sitting

than in the recumbent posture. In cases of anæmia, change of posture exercises a more remarkable influence upon the rapidity of the heart's action than in the healthy subject: and, as rapidity of the circulation is one of the conditions necessary for the development of the venous murmur, it follows that it ought to be more evident in the erect or sitting than in the recumbent posture. Besides, in the recumbent posture the fascia of the neck is more or less relaxed, and the vein is placed in an unfavourable state for the development of sound.

The arterial and venous murmurs will be again alluded to when I come to speak of what are termed "inorganic affections." It is only necessary here to observe, that these abnormal sounds are very common in chlorotic and anæmic subjects, and in individuals who have suffered considerable loss of blood; and they are heard in all cases where the blood is altered, its serous portion increased, and its viscosity lessened. We seldom or never examine a chlorotic subject in whom some of these sounds are not present, and the more advanced the stage of anæmia the more intense are the murmurs: as the anæmic condition is relieved by treatment, the sounds diminish in intensity, and eventually, when the patient's health is completely restored, they disappear.

## CHAPTER VII.

GENERAL SIGNS OF CARDIAC DISEASE.—COURTENANCE.—POSTURE OF THE PATIENT.—PALPITATION.—EPIGASTRIC PULSATION.—ANGINA PECTORIS.—THE PULSE AS A MEANS OF DIAGNOSIS.

THE general signs of the advanced stage of cardiac disease have considerable similarity, and the symptoms most generally present, are common to several diseased conditions of the heart. In the majority of cases, they have their origin in some impediment to the free passage of the blood through the left side of the organ, by which the circulation through the heart or through the lungs, or through both, is impeded.

Disease of the heart may lead to disease of the lungs, and *vice versa*. In a healthy condition of the parts, there is a perfect equilibrium between the development and the capacity of the two organs: the exact amount of blood is transmitted by the right ventricle to the lungs, which, in the latter organs, can be converted into arterial blood; and the exact amount of blood is returned by the pulmonary veins, which can pass freely through the left chambers of the heart. But, if this equilibrium is disturbed; if the pulmonary circulation is obstructed, owing to disease in the lungs, bronchial tubes, or pleura, the right auricle and ventricle become distended, and eventually, if the cause continues in operation, dilated or hypertrophied. If, again, there is an impediment to the free passage of the blood through the left side of the heart, congestion of the lungs as a natural consequence ensues; œdema follows, then hæmoptysis or pulmonary apoplexy. The effect of the obstruction of the pulmonary circulation extends to the right side of the heart; its cavities become distended, then dilated, or their walls become hypertrophied. The great venous trunks not being able to empty themselves, become distended, and the whole venous system congested: hence the lividity of the face and extremities, the congestion of the lungs, liver, and other ab-

dominal viscera, which is partly relieved by the escape of the serum of the blood into the cellular tissue of the extremities, giving rise to anasarca; or into that of the lungs, causing œdema pulmonum; partly by its effusion into the abdomen or thorax, constituting ascites or hydrothorax; and sometimes by the escape of the blood itself upon the congested mucous surfaces, giving rise to epistaxis, hæmoptysis, &c.; or into the pulmonary tissue, constituting the state with which we are familiar as pulmonary apoplexy.

The general symptoms of the advanced stage of cardiac disease are pretty constant; while those of its early stage vary as the disease is acute or chronic; they vary likewise according to its nature, and to the parts of the heart engaged. Thus, while in the early stage of the *chronic* forms of disease, there is scarcely anything characteristic in the symptoms, those which accompany the early stage of *acute* diseases of the organ are usually very well marked.

Among the general symptoms of heart disease, palpitation is usually one of the first which attracts attention. More or less dyspnœa is often present, which may or may not be accompanied by cough. The pulse, likewise, sometimes exhibits diagnostic characters. When the disease is more advanced, or when obstruction to the pulmonary or the general circulation has set in, other symptoms are usually superadded; signs of congestion of the lung, liver, kidney, spleen, or gastro-intestinal mucous membrane mark their appearance; orthopnœa supervenes upon the dyspnœa; and the over-loaded or over-distended vessels are relieved, in part, by the escape of the serum of the blood into the general cellular tissue, or into the serous cavities; or sometimes by the escape of the blood itself upon some of the mucous surfaces, or into the pulmonary tissue.

Before entering upon the consideration of these symptoms, it may be well to delay for a moment to consider whether there is anything in the expression or character of the countenance of a patient labouring under heart disease, or in the posture which he assumes, from which information as to its nature may be gathered. In the acute inflammatory affections of the investing or lining membrane of the heart, information may not unfrequently be obtained by attention to these particulars. In the chronic forms

disease nothing is to be learned from them as long as the disease is recent; but when the disease is advanced, they are sometimes sufficiently characteristic. Before the discovery of auscultation and percussion, and before diseases of the heart could consequently be diagnosed with certainty, these points received a much larger share of the attention of practitioners than at the present day: they are, however, of sufficient importance to deserve a short notice.

## COURTENANCE IN HEART DISEASE.

In the *acute* inflammatory affections of the lining or investing membrane of the heart, the countenance early acquires an anxious and depressed character; indeed, it is often so marked as immediately to attract attention; and, from it alone the practitioner has been induced to institute a minute examination into the condition of the central organ of the circulation, although the patient made no complaint of any symptom referable to it. This applies particularly to cases of acute rheumatism, in which the articular inflammation comes to be complicated with pericarditis, or endocarditis, or both. It is explained by the sympathy which subsists between the respiratory nerves of the face and the cardiac nerves. In affections of this nature, in infancy particularly, we commonly observe an elevation and depression of the *alæ nasi* in the respiratory act; movements depending upon the influence of the *portio dura* of the seventh pair (the respiratory nerve of the face, of Sir C. Bell), which are more marked in proportion as the respiration is more difficult. Indeed, in acute affections either of the lungs or heart in children, this is often a useful guide, and sometimes indicates a greater amount of disease than the general symptoms would lead us to suspect.

In the *chronic* forms of cardiac disease, little is to be learned at first, either from the expression of the countenance, or from the character of the individual features; in the advanced stage, however, the countenance becomes sometimes almost pathognomonic. Thus, in cases where the pulmonary circulation is much impeded, the venous system throughout the body becomes congested, the capillaries injected, the extremities swollen and cold, and the nails blue; the face becomes bloated and dusky, the eyelids puffed, the eyes staring, the conjunctiva suffused, the lips and

cheeks purple, the respiration laborious, the air passages loaded with mucus, and the jugular veins distended or pulsatile. On the other hand, it not unfrequently happens that in an equal advanced stage of some other form of cardiac disease, there is neither œdema or swelling of the extremities, the face instead of being puffed and dusky, is very pale and often expressive of pain. If dyspnoea occurs, it is in paroxysms, during which the respiration is gasping; there is a painful sense of constriction or impending dissolution, or the patient utters exclamations of pain which is referred to the præcordial region, or which shoots from the region of the heart to the spine or left scapula, and extends to the arm particularly the left.

Corvisart\* first made the remark, that in persons of the lymphatic temperament, the face instead of assuming a purple tinge becomes more pale than natural in the advanced stage of cardiac disease. Dr. Hope repeats the same remark: "In persons of the sanguine temperament (he observes), who are naturally florid, the lips and cheeks often become injected, and the countenance bloated while in individuals of the lymphatic temperament, who are naturally pale, it usually becomes still more so. Dr. Ormerod† attributes more influence to age than to temperament, and describes the character of the countenance in the advanced stage of valvular disease as being different in childhood, and adult age. Undoubtedly, age and temperament may modify the appearances in some respects; but, I do not think that either of them can be regarded as the cause of this difference, which appears to me to depend upon the *nature* of the organic lesion, and whether this has or has not occasioned *congestion* of the lungs. Thus, when it consists in a very contracted state of the mitral orifice, with hypertrophy and dilatation of the right ventricle, in which congestion of the lungs is a necessary result almost, the face will be congested and bloated, the lips and cheeks purple, and the lower extremities livid and anasarcaous. On the other hand, in cases of disease of the aortic valves permitting regurgitation into the left ventricle, the face is not bloated, the lips and cheeks are not purple, and the extremities are not livid or anasarcaous; but the face becomes pale, and the countenance anxious, and expressive of suffering.

When congestion of the lungs is combined with congestion

\* Essai sur les Maladies du Cœur.

† Gulstonian Lectures, Medical Gazette.



the *liver*, the complexion as Dr. Budd\* remarks, “becomes purplish, and at the same time *sallow*.” “As the blood, when its passage through the lungs is impeded, is imperfectly *decarbonized* and gives a purplish colour to the face, so, when its course through the liver is impeded, the blood is not completely freed from the principles of bile, and the countenance acquires a slightly jaundiced or *sallow* tint.” This had not escaped the observation of Dr. Bright, who remarks: “When obstruction takes place to the circulation through the chest, but more particularly when the heart becomes over-distended with blood, we observe the countenance gradually assume a dingy aspect, in which the purple suffusion of carbonized blood is mingled with the yellow tint of jaundice; the conjunctiva is more decidedly tinged, and if the disease continues long, sometimes completely prevails over the purple tint.”

*Prominence of the Eyes.*—A prominence of the eye-balls, giving the eyes a peculiar, staring appearance, as if they were larger than natural, or as if they were protruded forward, has been noticed in a few instances in connection with cardiac disease, or with symptoms simulating it, of which the most prominent was palpitation. It coincided in the majority of cases with an enlargement of the thyroid gland. The subjects were females, often of an hysterical habit; and the attacks of palpitation in some instances were prolonged and severe. In Dr. Parry’s posthumous work,† several cases are related under the head “enlargement of the thyroid gland, in connection with enlargement or palpitation of the heart;” the subjects were all females, and the majority were examples of organic disease of the heart; in only one, protrusion of the eyes is mentioned. Recently, Dr. Graves and Dr. Macdonnell have called attention to this phenomenon, and have alluded particularly to the prominence of the eye-balls. It appears to me to have a doubtful connection with cardiac disease. In one case, where the subject of it was an extern patient of the hospital, she had continued for several years much in the same state. In another, who presented the same peculiar appearance of the eyes, but without any enlargement of the thyroid body, and who died in hospital, a small tumor growing from the dura mater was found, on a post-mortem examination. In a third case, where the subject was also a female, about 25 years of age, and where

\* On Diseases of the Liver.

† Collections from unpublished writings, vol. ii.

protrusion of the eye-balls was also marked, and was complained of by the patient, which she said felt as if swollen, there was enlargement of the thyroid body; but neither palpitation or other symptom referable to the heart.

*Arcus Senilis*.—Another peculiarity connected with the eye, deserving of attention, is that which is familiar to us as the *arcus senilis*. Mr. Canton first demonstrated this to be the result of fatty degeneration of the cornea; and both he, and Dr. Williams have shown that it is sufficiently often met with in cases of fatty degeneration of the heart, to be entitled to be considered a diagnostic sign of this diseased state of the organ.

#### POSTURE OF THE PATIENT.

In the early stage of the chronic forms of cardiac disease nothing is to be learned either from the position of the patient or from the posture which he assumes. He is usually able to rest in the horizontal posture, and seldom finds greater difficulty in lying upon one side than the other; sometimes he prefers the supine posture: frequently it is with great reluctance that he remains in bed.

In acute affections, more particularly in that form of pericarditis where a large amount of fluid is *rapidly* effused into the pericardial sac, the patient is quite unable to lie down, or to obtain rest in the horizontal posture. Sometimes, the restlessness is so extreme that he cannot remain for more than a few minutes in any one position, and he is continually changing it; at others, he obtains partial relief only in one particular posture; any deviation from which is sure to add considerably to the dyspnoea and distress.

In the advanced stages of valvular disease, when the circulation through the lungs becomes greatly impeded, or when fluid is effused into one or both pleural cavities, the patient cannot lie down; he must have the head and chest elevated; or he is unable to remain in bed, and is forced to preserve the sitting posture. In extreme cases, he is often only able to snatch momentary rest by leaning forward and resting his head upon his knees, or upon a table. Sometimes the dyspnoea is so extreme, that he obtains ease only by lying on his face; this, however, is more frequently ob-

served in aneurism of the aorta pressing upon the trachea than in organic disease of the heart.

When, in addition, effusion to a considerable amount exists in the peritoneal cavity; or when the lower extremities have become much swollen, the position of the patient will be influenced in some measure by these complications. I have seen the dyspnoea aggravated in a remarkable degree by merely bringing the anasarca lower extremities to the same level with the body; and the patient could not rest unless they hung down by the side of the bed. In many such cases, indeed, the patient refuses to remain in bed, and sits constantly in a chair.

In other instances, where there is neither anasarca of the lower extremities nor ascites, but where pain or dyspnoea are the most prominent symptoms, and where the dyspnoea occurs in paroxysms of extreme severity, the position which the patient assumes in the paroxysm is peculiar: he sometimes seizes upon some fixed object against which he strongly presses the sternum; or he fixes the arms firmly, so as to call every muscle of inspiration into action; or he writhes about, and almost screams with agony. A patient was in hospital some time since, who during the paroxysms, was unable to obtain ease in any position but sitting upon the ground with the back firmly pressed against a wall. Dr. Hope has drawn a vivid picture of an aggravated case of this kind: "Incapable of lying down, the patient is seen for weeks, and even for months together, either reclining in the semi-erect position supported by pillows, or sitting with the trunk bent forwards and the elbows or forearms resting on the drawn-up knees." "With eyes widely expanded and starting, eyebrows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts round a hurried, distracted look of horror, of anguish, and of supplication; now imploring in plaintive moans, or quick, broken, accents and half-stifled voice, the assistance already often lavished in vain; and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death, to put a period to his sufferings."

## PALPITATION.

Among the general signs of cardiac disease, some may be said to be direct, others indirect: the former have obviously more value

than the latter. Among the direct symptoms palpitation is one of the most common; the character of the pulse being also occasionally diagnostic, may be included under this head: under it, also, turgescence of the jugular veins, pulsation in these veins, and fremitus cataire may be included: the latter have, however, already been considered. The indirect—or, as they are more correctly termed, the secondary symptoms—are common to diseases of the lungs as well as of the heart. Under this head may be included congestion of the lungs, cough, dyspnoea, orthopnoea, hæmoptysis, pulmonary apoplexy, congestion of the liver, spleen, kidneys, and brain, anasarca, and effusion into the cavities of the pleura and peritoneum.

Among the direct signs of cardiac disease, palpitation is perhaps the most constant and most frequent: it is often the first symptom which attracts the patient's attention; and it frequently distresses and harasses him throughout every stage of the disease. On the other hand, palpitation is frequently a prominent and troublesome symptom, when the heart itself is in a perfectly healthy state.

It has already been said that, in the healthy subject, where the chest is well formed and the heart and lungs have their normal proportions, the impulse of the apex of the heart is so slight that the individual is not sensible of it himself: the motion, in fact, is performed without his consciousness, and is only perceived in a very limited space when the hand is laid upon the parietes of the chest. Whenever, consequently, a person becomes sensible of the beating of his own heart, it may be said to constitute palpitation. Palpitation may therefore be defined: An unusual action of the heart, perceptible to the individual and inconvenient to him; the impulse being often stronger than natural, and the action of the organ either more rapid than in health, or intermittent, or irregular.

Palpitation presents numerous varieties; sometimes it is so slight that the patient is unconscious of it, or makes no complaint unless questioned; at other times, it is so violent as to elevate the clothes and shake the bed upon which he lies. Sometimes the impulse communicated is a slight, smart shock; sometimes it is strong, prolonged, and heaving, elevating the head of the observer; at other times the impulse is not altered, or is even weaker than natural. Sometimes the sounds of the heart are increased in inten-

sity, and, in rare cases, so much, that the patient can hear them himself when he lies upon the left side; at others, the sounds are not at all increased in intensity. In some cases palpitation sets in suddenly; in others, it makes its approaches very gradually. Sometimes the fits are short, quickly subside, and the patient is perfectly free from it in the intervals; at others, the fits are longer and more severe, or more or less palpitation is constantly present. Sometimes the palpitation is accompanied by uneasiness, by a sense of constriction, or of weight or pain in the region of the heart, or by a sense of sinking or of fluttering in the epigastrium; sometimes by dyspnoea or orthopnoea; sometimes by headache, vertigo, or noises in the head: at others, nothing of the kind is observed, and palpitation is the only symptom we are called upon to treat.

Palpitation, although one of the commonest symptoms of morbid states of the heart, frequently occurs independent of any organic change in this organ, and is not unfrequently a greater source of anxiety or alarm to the patient under such circumstances than in cases of organic disease. Thus it is a symptom in plethora; and in the very opposite condition, anæmia; it is a common attendant upon dyspepsia and derangement of the digestive organs, and it arises in debility from any cause: it is a frequent symptom in hysteria and hypochondriasis; it is not unusual in the early or advanced stage of pregnancy; and it accompanies several diseased states of the pulmonary organs.

The value of palpitation as a symptom, mainly depends upon whether it is *symptomatic* of diseased states of the heart, or whether it is *sympathetic* of some other affection: hence, it is of considerable importance that its cause should be determined. We shall therefore consider it here under two heads: viz., as it has its cause in organic disease of the central organ of the circulation, or as it occurs *independent* of cardiac disease.

*Palpitation depending upon Organic Disease of the Heart.*—Palpitation depending upon organic disease of the heart, usually, unless in cases of acute inflammation of the investing or lining membrane of the organ, comes on slowly and insidiously, and increases gradually; it is constant, although more distressing at one period than another; it is very generally combined with derangement of the general circulation, or with disorder of the res-

piration; and it is very generally also accompanied by well marked physical signs, or by other general symptoms of cardiac disease. Palpitation will therefore be a symptom in pericarditis and endocarditis, in hypertrophy, and in dilatation of the ventricles, or when these two states are combined; in diseased conditions of the valves or orifices of the left side of the heart, causing obstruction, or permitting regurgitation; in adhesion of the pericardium, the result of pericarditis; and in softening of the heart.

Thus, in acute pericarditis, or endocarditis, palpitation is often one of the first symptoms which attracts the attention of the patient or practitioner; the heart's action is more rapid than natural, and the impulse is increased. Cases, however, occasionally occur when palpitation is very slight, or entirely absent. Again, palpitation is one of the commonest symptoms of hypertrophy of the ventricles: here the impulse is increased, often considerably, but the action of the heart is not more rapid than in health. In dilatation of the ventricles, the palpitation is accompanied by increase in loudness of the heart's sounds, but the impulse has not much real strength. In the combination of these two states, or hypertrophy with dilatation of the ventricles, particularly when the disease is advanced, the palpitation is often very distressing, and the paroxysms are frequently prolonged and severe.

In organic disease of the valves or orifices of the left side of the heart, palpitation is very generally and constantly present; but as it is usually accompanied by hypertrophy, or dilatation of the ventricles, and as it always tends to produce these morbid states, the palpitation has its cause partly in the complication. Finally, in softening of the muscular tissue of the heart, palpitation is frequent; here, however, it is of the passive kind; the impulse is feeble, and the heart's action usually intermittent and irregular.

*Palpitation independent of Organic Disease of the Heart.*—Palpitation, independent of organic disease of the heart, is more common in the female than the male: it usually sets in suddenly, and is almost always intermittent. The impulse is often sharp and knocking, never heaving or prolonged, and the action of the heart is generally rapid. The palpitation occurs in paroxysms, often when the individual is in a state of repose: it is readily excited by mental emotion; and the physical and general signs of valvular or other disease of the heart are absent.

The palpitation which occurs under these circumstances presents several varieties, and may be traced to several causes, some of which are intrinsic to the heart, others are extrinsic to it. Thus, palpitation will ensue when the blood is returned to the heart more rapidly than in ordinary; or, when the motions of the heart are impeded, whether the exciting cause be seated in the lungs, in the parietes of the chest, or in the abdomen. Palpitation occurs in states of debility, whether this depends upon excessive evacuations, or arises in convalescence from acute disease; as well as in certain states of the nervous system, whether this be constitutional or acquired. Finally, it may ensue when two or more of the foregoing conditions are combined. Thus:

1. In a healthy subject, violent exercise on the one hand, or the indulgence in intoxicating liquors on the other, will hurry the circulation, and give rise to palpitation; but as soon as the exciting cause is removed, the effect ceases. In states of plethora, likewise, where blood is rapidly formed, and the vascular system becomes oppressed, more or less palpitation is generally present.

2. In cases where the bony parietes of the thorax are contracted and deformed; in adults whose chests are narrow and extremities long; or in young persons about puberty, whose growth has been rapid, palpitation is often complained of. Here its cause appears to lie in the narrowness of the chest, which interferes with the free play of the heart, or with the free expansion of the lungs. Palpitation is also occasionally complained of in the advanced stage of pregnancy, in cases of large ovarian or other abdominal tumors, or in ascites, in which the descent of the diaphragm is interfered with, or pressure is exercised upon the large vessels.

3. Palpitation is a very frequent symptom in states of debility; in anæmia, whether this arises from profuse hæmorrhage or accompanies amenorrhœa; or where profuse evacuations of any kind have occurred; under this head, sexual excesses and manustrupatio may be included. Palpitation likewise is a symptom of mercurial erithismus; and it not unfrequently arises during convalescence from acute diseases, particularly where mercury had been freely given; and after protracted fever it is by no means rare.

4. In persons of what is called the nervous temperament, palpitation is common, and is readily excited by mental emotion or by trifling causes. In hysteria, particularly in that form which



gets the name of spinal irritation, it is an ordinary symptom and in cases of hypochondriasis it is by no means unfrequent.

5. Palpitation is not unfrequently induced by excessive study by late hours, by the habitual use of strong tea, or the inordinate use of tobacco in the form of smoking ; by the suppression of some habitual discharge ; by prolonged mental excitement, whether of an exciting or depressing nature ; or by a sudden shock or fright. In these cases it partly depends upon derangement of the digestive organs, and partly upon debility, or constitutional or induced irritability.

Lastly, palpitation is frequent in persons of a gouty habit ; indeed, the first symptom indicating the approach of a fit is often palpitation, with fluttering and intermission of the heart's action. Palpitation is also a common accompaniment of dyspepsia ; here it is partly due to flatulence, which, by resisting the descent of the diaphragm, impedes the free expansion of the lungs, and interferes with the motions of the heart ; partly to the reflex sympathy between the parts concerned.

It not unfrequently happens in practice, however, that organic disease of the heart is associated with derangement of the digestive organs ; with an anæmic state of the system ; with a nervous, irritable, or gouty habit ; or with some of the other causes capable of giving rise to palpitation ; by which the difficulty of the diagnosis will be considerably increased.

In the following Table the more prominent characters of the palpitation, which depends upon organic disease of the heart, are contrasted with those of palpitation arising independent of disease of this organ :

**PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.**

1. More common in the male than the female.

2. Palpitation usually comes on slowly and gradually.

3. Palpitation constant, though more marked at one period than at another.

4. Impulse usually stronger than natural ; sometimes remarkably increased, heaving, and prolonged ; at others irregular and unequal.

**PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.**

1. More common in the female than the male.

2. Palpitation usually sets in suddenly.

3. Palpitation not constant, having perfect intermissions.

4. Impulse neither heaving or prolonged ; often abrupt, knocking, and circumscribed, and accompanied by a fluttering sensation in the præcordial region or epigastrium.



5. Percussion elicits a dull sound over an increased surface, and the degree of dulness is greater than natural.

6. Palpitation often accompanied by the auscultatory signs of diseased valves.

7. Rhythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.

8. Palpitation often not much complained of by the patient; occasionally attended by severe pain, extending to the left shoulder and arm.

9. Lips and cheeks often livid; countenance congested; anasarca of lower extremities common.

10. Palpitation increased by exercise, by stimulants and tonics, &c.; relieved by rest, and frequently, also, by local or general bleeding, and an antiphlogistic regimen.

5. Extent of surface in the region of the heart, which yields naturally a dull sound on percussion—not increased.

6. Auscultatory signs of diseased valves absent; bruit de soufflet often present in the large arteries, and a continuous murmur in the veins.

7. Rhythm of heart usually regular; sometimes intermittent; its action generally more rapid than natural.

8. Palpitation often much complained of by the patient; readily induced by mental emotion, and frequently accompanied by pain in the left side.

9. Lips and cheeks never livid; countenance often chlorotic; anasarca absent, except in extreme cases.

10. Palpitation increased by sedentary occupations; by local and general bleeding, &c.; relieved by moderate exercise, and by stimulants or tonics, particularly the preparations of iron.

*Immediate cause of Palpitation*.—The blood which circulates through the cavities of the heart, is considered to be the natural stimulus to the contractions of the organ: thus, in a state of health, when this fluid is returned to the right side of the heart more quickly than in ordinary, its action becomes more rapid. In this way exercise increases the frequency of the heart's contractions, and hurries the circulation, which returns to its normal frequency after the patient has remained at rest for a time.

In states of plethora, in which blood is rapidly formed, palpitation we have seen to be a common symptom; there is here an increased amount of blood in the vessels, and this fluid is of a more stimulant nature, perhaps; the palpitation which ensues in these cases is not accompanied by quickened action of the heart. In the very opposite condition to this—viz., anæmia—where the blood is poor and watery, and deficient frequently in quantity, palpitation is also extremely frequent, and the heart's action is more rapid than in health: here the heart appears to contract more frequently, to compensate for the deficiency in the quality of the blood.

Again, when the valves or orifices of the heart are diseased, and the passage of the blood out of, or through its chambers is im-

peded, palpitation ensues, and the organ is excited to increased action in order to overcome the obstacle to the transmission of the blood. Palpitation is, however, by no means rare, independent of any of the foregoing circumstances; in such cases, the nervous system is probably at fault, though the exact mode in which this causes palpitation is not quite clear. When we come to speak of functional derangement of the organ, we shall have occasion to return to this point.

Before dismissing the subject of palpitation, I may observe that this symptom frequently is present in a marked degree in a class of patients who present themselves at hospital, without their appearing to be conscious of it. When questioned, they all admit that they feel some oppression in the præcordial region, but hardly admit that it amounts to palpitation, though, when we come to examine the chest, the action of the heart is often much increased and its impulse so strong as to raise the head of the observer. This, no doubt, arises in some measure from the sensibility being blunted by the ill oxygenation of the blood; and in some measure also, from the parts having had time to accommodate themselves to their altered condition. On the other hand, it is no less remarkable, how often, when palpitation depends simply upon functional derangement, the patient's attention is directed mainly to it; he is most unhappy in consequence, and can scarcely be made to believe that he is not the subject of organic, and, in his opinion therefore, of incurable disease of the heart.

#### EPIGASTRIC PULSATION.

A pulsation in the epigastric region which is evident to the eye, and perceptible to the hand, and of which the patient himself is conscious, is by no means uncommon; it is occasionally symptomatic of organic disease of the heart, or of some part of the arterial system; sometimes it is a sign of simple functional derangement; and at others it is independent of either.

In describing the normal position of the heart, we saw that this organ rests upon the cordiform tendon of the diaphragm; now if the sternum is short and the heart's action strong, there is more difficulty in accounting for the heart's impulse being seen and felt in the epigastrium under such circumstances, than for an impulse being communicated to the abdominal parietes, by the act of coughing.

*Epigastric Pulsation in Emphysema.*—When the volume of the lungs is increased, as in emphysema of these organs, the heart will be pushed somewhat downwards, and its pulsation will be evident in the epigastric region ; and as this condition of the lungs is in general associated with enlargement of the right side of the heart, particularly when it has persisted long, the pulsation is usually well marked. This form of epigastric pulsation is observed high up in this region, it is quite perceptible to the hand, and it is evidently produced by the heart itself. It occasions less distress than some of the other forms, and we are not often called upon to treat it alone.

*Pulsation the result of Congestion.*—When hepatic is combined with gastro-intestinal congestion—the result of impediment to the return of the venous blood to the right side of the heart—this may react upon the arteries which supply these viscera, and through the celiac axis, and superior and inferior mesenteric arteries upon the abdominal aorta ; the pulsation of this vessel becomes more distinct, and stronger, constituting one form of epigastric pulsation, and resembling that described by Dr. Faussett,\* which was referred by him to local or visceral congestion, or to sub-acute inflammation. This variety of epigastric pulsation is accompanied by “pain on pressure at the epigastric region, or towards the umbilicus, with considerable fulness at the part ; the digestive organs are deranged, the bowels torpid, and the feet cold. The pulsation is most severe towards the afternoon, or soon after dinner, and is accompanied by a sense of vital depression. On auscultation, a loud whizzing sound is heard ;” we may, however, have this form of epigastric pulsation without any murmur being audible on auscultation.

A pulsation of the abdominal aorta, or its immediate branches, which is symptomatic of inflammatory disease in the abdomen has been noticed by Dr. Stokes.† It consists in “a throbbing generally commensurate with the disease, removed by treatment calculated to relieve enteric inflammation, and aggravated by every thing which increases it.” “In other words, we may have (he observes) from enteritis or peritonitis a throbbing of the abdominal aorta, or its branches, perfectly analogous to the morbid action of the radial artery in whitlow, or of the carotids or temporal arteries

\* Dublin Journal of Medicine, vol. ii.

† Ibid, vol. v.

in cerebritis." The diseases in which this pulsation was observed were gastro-enteric fever, peritonitis, and fever consecutive to corrosive poisoning.

*Epigastric Pulsation in adhesion of the Pericardium.*—Pulsation in the epigastric region was one of the earliest mentioned diagnostic signs of adherent pericardium; Mr. Burns\* considers it to be a constant symptom; "when the ventricles contract they drag the pericardium with them (he observes), and the pericardium again pulls the diaphragm after it, so that by the alternate elevation and depression of this septum, the chief pulsation comes to be referred to the epigastric region." The pulsation in these cases is rather an undulatory movement of the parietes than a distinct pulsation; it is perceived high up in the epigastric region, but it has no real strength, for when the hand is laid upon the part, or when pressure is made, no impulse is communicated.

*Nervous and Hysterical Pulsation.*—A form of epigastric pulsation, observed in subjects in whom the heart is perfectly healthy, occurs occasionally in nervous and hysteric females; which is nothing more than the pulsation of the abdominal aorta communicated to the parts resting on it, owing to temporarily increased action of the heart. The pulsation is most marked below the epigastrium, and the impulse communicated to the hand is a short, smart shock, very unlike that of aneurism. On auscultation, a short whiff is audible, particularly when the stethoscope is pressed firmly upon the part. This variety of epigastric pulsation has remissions or complete intermissions; it is excited or aggravated by mental emotion, deranged digestion, or intemperance; and is sometimes a source of considerable distress to the patient.

*Anæmic Pulsation.*—A pulsation in the epigastric region communicated by the abdominal aorta, occurs likewise in anæmic subjects, particularly when this state has been brought on by profuse hæmorrhage. The pulsation is strong, abrupt and knocking, accompanied by a short, whizzing murmur, leading, sometimes, to the suspicion of aneurism. The pulsation, which is evident to the eye as well as perceptible to the hand, is most marked below the epigastrium, and is in a great measure limited to the line of the artery. In such cases, visible pulsation is present, and a

\* Treatise on Diseases of the Heart.

similar sound on auscultation is audible in the large vessels which come off from the arch of the aorta.

*Pulsation communicated to abdominal Tumors.*—Diseased states of some of the contents of the abdomen, as of the omentum, mesentery, or pylorus; or air or fœces confined in the transverse colon, occasionally have an impulse communicated to them, where they rest upon the aorta below the diaphragm. The pulsation is both evident to the eye and perceptible to the hand, and has been mistaken for abdominal aneurism. It may be distinguished by the impulse wanting the heaving character of the impulse of aneurism in this situation, and by the absence of lateral impulse; by no murmur being audible on auscultation, or by its different character; by the mobility of the tumor, by the previous history of the case, and by the absence of pain, and other signs of abdominal aneurism.

Dr. Baillie,\* many years since, called attention to a variety of epigastric pulsation of which he had met several examples, and which probably belongs to one of the forms already noticed. He observed it in persons about the middle period of life, most frequently in the male; the pulsation which was often visible to the eye, was stronger in some subjects than in others; varied in strength at different periods, and was usually most marked in the horizontal posture. He believed it to be connected with deranged digestion and an irritable constitution. It is apt (he observes) to cause a good deal of unnecessary anxiety in the patient; it does not depend upon any diseased condition of the artery, for he has known it to continue well marked during twenty-five years without the health suffering; and in two instances in which he made a post-mortem examination, the artery was perfectly healthy.

## ANGINA PECTORIS.

Among the secondary symptoms of cardiac disease, one occasionally met with is familiar, by name at least, to practitioners, as "*angina pectoris*." It presents itself in several degrees, sometimes in a mild, at others in a very intense form; but, whether mild or severe, its prominent feature is pain, and pain of a peculiar kind.

\* Trans. of College of Physicians, vol. 4.

A paroxysm of angina, when well marked, is characterised by sudden, severe, constrictory or burning pain, referred to the lower part of the sternum, or to the præcordial region, extending through the chest to the left scapula and up the sternum to the root of the neck, which compels the patient if walking, instantly to stop, and almost prevents inspiration. Pain is felt likewise in the left shoulder, which reaches to the elbow, rarely to the hand, often with a sensation of numbness in the parts; accompanied by a feeling of constriction of the chest, by a tendency to syncope, or by intense anxiety and a sensation of approaching dissolution, with or without palpitation, or irregular action of the heart.

The paroxysm of angina varies in length and intensity; it occurs at irregular intervals, but is generally brought on by muscular exertion of some kind, as running or walking quickly up a hill, particularly after a full meal; or by mental emotion, as a fit of passion; and it is most common in persons of a gouty habit, who have passed the meridian of life; and in males rather than females. In many instances angina occurs in a mild form, the patient suffers merely from constrictory pain in the præcordial region on walking quickly, or ascending a height, which lasts but a few seconds and then passes off. In others, however, the paroxysms are more intense and of longer duration, they occur more frequently; while they are excited by more trifling circumstances, such as the simple acts of dressing or undressing, passing from a warm to a colder atmosphere; or, they may supervene during sleep.

*Causes of Angina.*—In the generality of treatises on diseases of the heart, angina pectoris is classed apart as a distinct disease. The earlier writers upon the subject, supposed it to have some mysterious connexion with ossification of the coronary arteries; while others referred it rather to excessive disposition of fat upon the heart. Modern pathologists having failed not unfrequently in discovering either of these morbid changes, and seeing the suddenness with which it supervenes and subsides, refer it to “*spasm of the heart.*” This is the view taken of it by Dr. Latham; Dr. Chever’s\* definition is however more precise, he regards it as “*a spasm of a weakened heart.*” Other pathologists again, looking more to the character and intensity of the pain,

\* Pract. Treatise on Diseases of the Heart, Calcutta, 1851.

regard it as a form of neuralgia, and describe it under the title "*neuralgia of the heart.*"

I do not think sufficient evidence has been adduced to entitle angina pectoris to be regarded as a distinct disease; neither do I think it necessary to call in the aid of spasm or neuralgia, when there are circumstances in its clinical history and pathology which seem to be quite capable of explaining all its phenomena independent of either. Thus, angina, in a well-marked form, is not seen except in cases of organic disease of the heart; and as a general rule, it is not observed even in these, unless something occurs either to disturb the action of the heart or to hurry the circulation.

The *immediate cause* of angina pectoris appears to me to be a sudden impediment to the *coronary* circulation, particularly to the return of the blood by the coronary veins; itself, in general, the result of a temporarily over-distended state of the chambers of the heart, and an inability in them to empty themselves, whether owing to weakness of the muscular tissue of the parietes of the left ventricle, or to other causes. For instance, if the cavity of the left ventricle is considerably dilated, or its walls are attenuated or softened, or have undergone fatty degeneration, the contractile power of its muscular tissue will be impaired in proportion; and if the circulation happens to be suddenly hurried, or the heart's action to be suddenly disturbed, the cavity of this ventricle might become so much distended as to render it incapable of contracting upon its contents, which would be quickly followed by distension of the auricle on that side, and, if relief is not soon experienced, by distension of the right side of the heart.

It is scarcely necessary to say, that in a normal state of the circulation all the chambers of the heart are never full of blood at the same moment. When the ventricles are filled, the auricles are comparatively unfilled, and *vice versa*. Here, however, we would have a suddenly distended state of the chambers on both sides of the heart at the same moment. From the position of the coronary vessels in the grooves of the heart, they cannot, we know, suffer compression during the alternate movements of the ventricles and auricles; but if the auricle and ventricle upon each side of the heart are distended at the same time, these vessels, but particularly the veins, must suffer compression, by which their circulation will be impeded; and the great coronary vein, in addition,



may be prevented from freely emptying its contents into the right auricle, in consequence of the distended state of this cavity.

We know, likewise, that the normal capacity of the pericardial sac is but little greater than that of the heart in its ordinary state of distension; and that this membrane is composed of tissue which does not *suddenly* yield. The parietes of the heart would therefore, under the circumstances that I have described, be placed between two compressing forces—an undue amount of blood in the chambers of the organ, and the unyielding pericardium upon the outside. The effect of this compression of the heart's tissue would necessarily be to impede still further the coronary circulation, and to clog still more the movements of the organ; and the condition of the heart would be somewhat analogous to that in which a large amount of fluid was *suddenly* effused into the pericardial sac, from rupture of a vessel or other cause.

The organic lesions of the heart most likely to be attended with angina would therefore be, a condition of the aortic valves permitting free regurgitation, with a rigid, dilated state of the ascending portion of the arch of the aorta, which permits the blood from the large vessels to regurgitate into it, combined with, either

1. Dilatation of the cavity of the left ventricle; or,
2. Attenuation of the parietes of the left ventricle; or,
3. Softening or fatty degeneration of the muscular tissue of this ventricle.

For instance, when the aortic valves permit free regurgitation there is, at each ventricular diastole, a reflux of blood from the aorta into the ventricle at the same time that the current is entering it from the auricle; the left ventricle consequently is unable to empty itself, the state of distension in which it is kept leads to permanent dilatation of its cavity, which tends still further to embarrass the circulation by enfeebling the ventricle, and diminishing its power of expelling its contents.

It has been shown by Hales, that "each square inch of the surface of the interior of the ventricles has a pressure upon it during the systole equal to about four pounds;" and "as the resistance which the heart has to overcome in contracting is, according to hydrostatic laws, in proportion to the extent of the inner surface of the cavity at the commencement of the systole, if the cavity of the ventricle is dilated, more force will be requi-



to enable the ventricle to expel its contents ; and the greater the amount of the dilatation the less able will the ventricle be to overcome the resistance. In such a state of the aortic valves, and in such a condition of the ventricular cavity, if the parietes of the left ventricle are, in addition, attenuated or softened, the ventricle will be still less capable of overcoming the resistance, and a very trifling muscular exertion, or a sudden mental emotion, may lead to over distension of its cavity, followed by distension of the other chambers of the heart. If, at the same time, the arch of the aorta is dilated, and its coats are rigid and inelastic, permitting the blood from the large arteries which come off from the arch, to regurgitate into it, the coronary circulation will be necessarily impeded, and a paroxysm of angina may be the result.

Any one of the foregoing morbid conditions of the heart may be present, or two or more of them may be combined, without angina necessarily occurring ; indeed, as long as the circulation continues tranquil, and as long as the left ventricle is able to get rid of the blood which enters its cavity, the latter cannot become over-distended. If, however, the heart's action is disturbed by some sudden mental emotion, or other cause ; or even without this occurring, if the stomach is loaded with indigestible food, and it and the intestines are distended with flatus, by which the cavity of the chest is encroached upon, and the heart's movements are impeded, a paroxysm of angina may be the result. Hence, in persons who have had previous attacks, it is liable to supervene during sleep, when it may be the result of a frightful dream, disturbing the heart's action ; or, of considerable distension of the stomach by flatus, impeding the movements of the organ.

Dr. Forbes,\* in his able article on the subject, has shown that *plethora* is a very common complication of angina ; a state which, if combined with a weak heart, would give a further predisposition to the attack. Thus, "the subjects of angina (Dr. Forbes observes) are mostly of the male sex, above 50 years of age, and a great majority of them belong to that class of persons who are enabled to indulge in full living, without the necessity of undergoing severe bodily labour." Again, "gout is a very frequent disease in persons subject to angina, and obesity is extremely common." "The very existence, too, of angina tends (he adds)

\* Cyclopædia of Practical Medicine.

to produce plethora if it did not previously exist; a sedentary life and abandonment of all active bodily exertions, are almost inevitable consequences of the disease."

It may, perhaps, be objected that the foregoing explanation of the cause of angina is insufficient to account for the peculiar pain which accompanies a paroxysm. I cannot, however, imagine any state more likely to be attended by intense distress, anxiety, and suffering, with a sensation of impending dissolution, than such as I have described—where the chambers of the heart are immoderately distended, the coronary circulation temporarily obstructed, the heart's movements clogged or impeded, or its muscular tissue compressed. It has been objected to this theory, that "in cases of emphysema with bronchitis, where the heart is greatly congested, and usually becomes enlarged and softened, angina hardly ever occurs." But the injurious effects of emphysema and chronic bronchitis are exerted upon the *right* not the left side of the heart, and over-distension of its cavities is relieved by the provision which permits of regurgitation through the tricuspid orifice whenever over-distension of the right ventricle occurs.

Angina pectoris, in its most marked form, is almost peculiar to advanced life; thus, of 84 cases recorded by Dr. Forbes, 72 were above 50 years of age, and only 12 under that age. The reason of this is sufficiently obvious; the morbid conditions upon which it depends are, in some measure, limited to advanced life. Again, when a person has been once the subject of angina, subsequent attacks are very likely to ensue if the exciting causes come into operation, because the diseased states on which it depends are irremediable. Lastly, angina is rare in the female compared with the male; because the diseased state of the aorta so frequently associated with it, is very seldom met with in the female; indeed, regurgitant disease of the aortic valves, itself, is less frequent in the female than the male.

In conclusion then, I would say, that angina pectoris ought to be regarded rather as a *symptom* of organic disease of the heart than as a distinct form of disease; in fact, what dyspnoea is to the lungs, angina appears to be to the heart; and it might without impropriety be termed the *dyspnoea of the heart*. Thus both are met with in very variable degrees of intensity; both have some-

times apparently almost purely a spasmodic character, and both are often the result of mechanical causes—in the one case, of some impediment to the free passage of the air into or out of the lungs; in the other, of an impediment to the circulation in and through the heart. It would indeed, in my mind, be almost as absurd to class dyspnoea apart, and describe it as a distinct affection, as it is to make angina a separate disease. As dyspnoea may arise under variable and opposite states of pulmonary disease, so angina may ensue in different forms of cardiac disease. We can, likewise, easily understand from what precedes, that if the paroxysm of angina is slight, it may pass off spontaneously; or under the influence of stimulants and other appropriate measures, the heart may be enabled to get rid of the blood which distends its cavities; while, if the paroxysm is severe and continued, complete arrest of the coronary circulation may ensue, followed necessarily by cessation of the heart's action, and the death of the patient.

#### *Conclusions respecting Angina Pectoris.*

The conclusions which I would draw from what precedes are,

1. That angina pectoris is to be regarded as a symptom of disease of the heart, not as a distinct affection.
2. That it does not occur except where organic disease of the heart, generally of long standing, exists.
3. That its connexion with spasm, or neuralgia, is doubtful.
4. That its probable cause lies in impediment to the coronary circulation, particularly to the return of the blood by the coronary veins.
5. That the diseased states of the heart in which it is most liable to ensue, are a condition of the aortic valves permitting free regurgitation, with a rigid dilated state of the ascending portion of the arch of the aorta; combined with, either dilatation of the cavity, or attenuation or softening of the parietes of the left ventricle.
6. That even in these diseased states, angina may not occur unless the heart's action is suddenly disturbed, or its movements are clogged, or impeded by some mechanical cause.

## THE PULSE AS A MEANS OF DIAGNOSIS IN CARDIAC DISEASE.

Previous to, and even for some time subsequent to the discovery of auscultation, the pulse as an aid to diagnosis in cardiac disease, was of very little value ; indeed, it is obvious that until the order of succession of the movements of the heart was known, and the auscultatory signs which characterise the several diseased states of the valves were laid down with some precision, any information derived from the characters of the pulse must have been very vague. The obscurity which formerly invested this subject has, however, now been in a great measure dispelled ; and the labours of successive investigators have shown, that certain forms of cardiac disease are capable of impressing peculiar and well-marked characters upon the pulse.

The pulse is produced by the contraction of the left ventricle, which at each systole propels a certain amount of blood into the aorta ; “the blood not being able to escape from the arteries as quickly as it is forced into them by the ventricle, on account of the resistance it experiences in the capillaries, necessarily exerts a pressure on the elastic coats, and thus gives rise to the pulse.” The pulse, therefore, corresponds with the ventricular systole, and with the first sound of the heart ; and when the heart, the arterial system, and the blood, are each in a normal condition, the force, the strength, the frequency, and the fullness of the radial pulse, may be taken as a measure of the strength or feebleness of the systole of the left ventricle—of the rapidity with which the movements of the heart are performed, and of the amount of blood transmitted at each systole of the left ventricle.

At each systole of the left ventricle the arterial tubes become elongated ; hence, in situations where these vessels have naturally a curve, this curve is increased ; and where they are naturally straight, they become slightly curved ; when the ventricular systole ceases, they return to their former position. In thin subjects this locomotive movement is perceptible in vessels near the surface, as in the temporal artery and in the radial at the wrist ; and this which is in reality a natural phenomenon is not unfrequently set down as a sign of disease. It, undoubtedly, becomes more marked in cases where regurgitation through the aortic valves, or into the aorta itself occurs ; but a visible and locomotive pulse in these

arteries, unless accompanied by other signs of disease, has no value by itself, and is frequently observed in cases where the heart and arterial system are healthy.

At the moment that the pulse is felt, the arteries, in addition to becoming elongated, dilate, and increase in volume. "Every fluid exposed to pressure from behind," Weber\* observes, "reacts on the surrounding parts, not merely in the line of direction of that pressure, but on every side; and, therefore, at each contraction of the ventricle, the blood the arteries contain, reacts in consequence of the impulse pressure of the injected blood, not merely longitudinally, but laterally, the arteries being fixed at both extremities." It has been denied by some physiologists that any dilatation of the arteries occurs: it does, however, take place; and M. Poiseuille,† by means of an apparatus, capable of being applied round an artery, has determined the amount of this increase to be, for the larger arteries about the one-eleventh of their diameter. The increased capacity of the arterial tubes depends more, however, upon their elongation than upon their dilatation.

The beat of the pulse in the radial artery is a little later than the ventricular systole; but the interval is almost inappreciable, unless the pulse is unusually slow: in arteries further from the centre of the circulation this is somewhat more marked. Thus, according to Weber, the beat of the anterior tibial artery upon the dorsum of the foot, is between the sixth and the seventh part of a second later than that of the axillary artery. According to Hamersyk,‡ the pulse at the wrist, in the temporal artery, and in the femoral, near Poupart's ligament, is perfectly synchronous; and according to Weber, the pulse of the axillary artery, and of the external maxillary artery, is synchronous. The radial pulse is said to follow the ventricular systole by a more appreciable interval when the aortic valves permit regurgitation: if it really does so, it is not a sign of any importance. "In debilitated and atonic states of the system the radial pulse," Dr. Williams§ observes, "follows the first sound of the heart by a distinct interval, which is occupied by the transmission of the wave along the course of the comparatively lax and yielding artery."

In some individuals, to all appearance in perfect health, the pulse

\* Quoted in Dublin Journal.

† Magendie's Journal.

‡ Gazette Médicale. March, 1844.

§ On Diseases of the Chest.

is preternaturally slow ; in others, it is preternaturally quick ; and in others, again, it is intermittent. These peculiarities are connected with a similar condition of the heart's action ; they appear to be the result of idiosyncrasy, and are sometimes hereditary. The curious fact has been occasionally noticed, that in an individual whose pulse in health intermitted, it became regular on the invasion of illness of a febrile character. It is important to bear in mind that these peculiarities in the pulse are occasionally met with, lest they should be too hastily set down to disease of the heart, with which they may have no connexion. Disease of the right side of the heart exercises no direct influence upon the pulse, but valuable information may sometimes be obtained from its characters in diseased states of the left side of the organ. Although the radial pulse is, in the great majority of cases, an indication of the frequency of the contractions of the left ventricle, it does not necessarily indicate the force of the contraction. Thus, in some cases of disease, although the action of the heart is violent, and its impulse strong, the radial pulse is small and feeble ; while, on the other hand, we know that in cases of local inflammation, the artery going to an inflamed part may beat very strongly, although the heart's action is not increased. In all doubtful cases, therefore it is advisable to place one hand upon the præcordial region, or to auscultate the region of the heart, while the finger is on the radial pulse. “ We can thus,” Dr. Williams observes, “ take a far more accurate survey of the condition of the circulation, than by examining these parts separately ; and the utility of this method is by no means confined to affections of the heart.”

In several morbid conditions of the heart, the pulse becomes quicker than natural : I am not aware of any diseased state of the organ in which it necessarily becomes slower, though this condition of the pulse is occasionally observed in softening of the muscular tissue of the organ, more rarely in very contracted states of the aortic orifice. The pulse, in some diseased states of the heart, becomes stronger, fuller, and harder than natural ; in others, softer, weaker, smaller, or feebler than natural ; and in others, again, it becomes jerking and receding. In all these cases the pulse may be regular ; but it not unfrequently happens that its rhythm is disturbed, when the pulse intermits, or becomes unequal or irregular ; and these conditions of the pulse may be either

combined together, or with some of the other states mentioned above.

The morbid conditions under which these varieties of the pulse occur, may consist in inflammation of the investing or lining membrane of the heart; in alterations of its muscular tissue; in changes in the capacity of its cavities; in diseased conditions of its valves or orifices; in alterations of the blood; in diseased states of the arteries; and in derangement or disease of other organs reacting upon the heart.

*The quality of the blood* contained in the vessels exerts a material influence upon the pulse; when this fluid is attenuated, when its red particles are diminished, its serous part increased, and its viscosity lessened, whether this depends upon profuse hæmorrhage, or arises under other circumstances, the contractions of the heart become more frequent, and the radial pulse is consequently quickened; at the same time, the blood is impelled with less force; while the healthy tension of the arteries being diminished, and their tubes not being filled, the pulse acquires a jerking character resembling the pulse of aortic regurgitation.

*The quantity of the blood* contained in the vessels, no less than its quality, exercises some influence upon the pulse. “When there is a due correspondence between the capacity of the vascular system, and the quantity of blood circulating in this system, the coats of the arteries will (Dr. Copland observes\*) be kept in that state of healthy tension, or tone, favourable to a regular, firm, free, natural, or healthy pulse, varying chiefly in frequency with the state of the heart’s action.” When the amount of blood in the system is greater than natural—in other words, in that state of the system denominated plethora—the pulse is full and hard, but not increased in frequency. The sensation produced by an artery in cases of excessive vascular fulness, Dr. Copland designates by the term “oppression;” the vessel feeling as if “it were kept in a state of tension, or of distension, in the interval between the beats.”

*Pulse in hypertrophy of the left Ventricle.*—When the parietes of the left ventricle are increased in thickness—in other words, in hypertrophy of the ventricle—its systole will be strong in proportion, the blood will be propelled into the aorta with increased force, and the radial pulse will be strong and hard: its velocity

\* Dict. of Pract. Medicine.



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\* Dict. of Pract. Medicine.

will not, however, be increased ; but, as the systole of an hypertrophied ventricle takes a longer time to be completed, the pulse will “dwell longer under the finger.” When some dilatation of the cavity of the ventricle is combined with hypertrophy of its walls, the pulse, as long as the circulation continues free, will have the same character, and, in addition, it will be full, because a larger amount of blood will then be impelled at each systole.

*Pulse in dilatation of the left Ventricle.*—When the cavity of the left ventricle is dilated, and its walls are attenuated, or even though they may preserve their normal thickness, the ventricular systole will be performed with less vigour, and the blood will be propelled with less force than in a healthy state of the heart : hence the radial pulse will have nearly opposite characters to those of the former state ; and, instead of being hard and strong, and dwelling long under the finger, it will be soft and weak, though it may be large.

*Pulse in aortic regurgitation.*—When the aortic orifice, or its valves, become altered by disease, or when, from any other cause, the valves imperfectly fulfil their function, the blood will regurgitate into the left ventricle at each diastole, and the pulse in consequence will acquire a peculiar character, being jerking and receding, though regular, while the pulsation of the arteries of the upper extremities and neck is visible and locomotive. This, which is sometimes termed the pulse of unfilled arteries, is very peculiar, and when once felt can scarcely be mistaken. In well-marked examples, it appears as if the blood was divided into separate little balls, which pass in rapid succession under the finger. This sensation becomes more marked if we apply the finger to an artery of larger calibre than the radial, as the brachial ; and if we lay two or more fingers upon the line of the artery.

*Cause of the jerking pulse of aortic regurgitation.*—The explanation of the cause of this peculiarity in the pulse is sufficiently simple. We have seen that the arteries, at the moment that the ventricular systole occurs, dilate ; when the diastole occurs they return to their former state, owing to the elasticity of their coats. We have seen, also, that the arteries in the healthy state are always filled : there is a continuous column of blood in them ; and the fresh portion transmitted into the aorta at each contraction of the left ventricle, displaces a column of blood of equal

size; an impulse is communicated, and the pulse is felt. Thus when an artery is wounded, or divided across, the blood escapes in a continuous stream, but is accelerated at each ventricular systole; the continuous stream being produced by the elasticity of the artery reacting upon the blood, in the intervals between the systole of the ventricle. Now, when the aortic valves imperfectly fulfil their office, the moment that the ventricular systole ceases a portion of the blood returns from the aorta into the left ventricle, there is a backward motion of the blood in the aorta as the ventricle dilates, which may be favoured by the elasticity of the coats of the vessel. The arteries of the upper extremities and neck of course feel the effect most; they are less perfectly filled, the healthy state of tension of their coats is diminished, they react with less force upon their contents, and the blood has a forward motion, or, at least, is propelled with any force only during the period that the ventricular systole lasts: hence the column of blood appears to be interrupted, and the pulse has this jerking and receding character.

In cases of anæmia, the same effect seems to be produced by the coats of the arteries losing tone or elasticity in consequence of deficient nutriment: the vessels are consequently imperfectly filled, and the blood is moved forward apparently only at the period of the ventricular systole. This peculiarity of the pulse is not however limited to the cases mentioned, it is observed sometimes in aneurism of the ascending or transverse portion of the arch of the aorta; as well as in cases of disease of the aorta itself, when this vessel has become rigid and elastic from adventitious deposit, which condition is often accompanied by dilatation of the artery. Here it is produced by the regurgitation of the blood from the large arteries, during the ventricular diastole, into the aorta itself, or into an aneurismal sac.

In addition, in patency of the aortic valves, the pulsation of the superficial arteries of the head, face, and upper extremities, is visible and locomotive; as first pointed out by Dr. Corrigan.\* This phenomenon is most remarkable in the temporal and radial arteries and their branches, in which "the arteries sometimes (as Dr. Williams observes) appear like worms under the skin, wriggling into tortuous lines at each pulse." Delay of the radial pulse

\* Edinb. Med. and Surg. Journal, vol. xxxvii., 1832.

has been set down as a symptom of patency of the aortic valves; but, as Dr. Douglas\* observes, "it has a doubtful connexion with valvular disease; and it does not occur except in conjunction with more or less dilatation." He says he has met with it altogether apart from valvular disease.

The radial pulse, in the majority of cases is an index of the strength, as well as of the frequency of the ventricular systole; in nervous palpitation, however, although the impulse of the heart may be strong the pulse is weak, but as the palpitation subsides it becomes fuller; again, it may happen that every contraction of the left ventricle is not propagated to the radial artery, owing to its systole being too feeble to propel the blood with sufficient force to give an impulse capable of being felt with the finger. If this recurs with regularity at every second beat, the pulse will appear to be preternaturally slow; and this, no doubt, was the condition in some of the cases of remarkably slow pulse reported previous to the discovery of auscultation. The error will be corrected by laying the stethoscope upon the præcordial region, while the finger is upon the pulse.

### *Intermission and Irregularity of the Pulse.*

The rhythm of the heart and the regularity of the contraction of the ventricles, are in general manifested by the pulse at the wrist: the heart's action cannot be intermittent or irregular without its being communicated to the radial artery; but the pulse may intermit, although the heart contracts regularly, if every fifth, sixth, or seventh ventricular systole, as the case may be, is too feeble to propel a sufficient quantity of blood to communicate an impulse to the radial artery.

*Intermission* may be regarded as the slightest degree of derangement of the heart's action. It is not uncommon, as has already been observed, in individuals in whom the heart and arterial system are healthy; the intermissions may occur at regular or irregular intervals, and this state may continue through life. This form of derangement of the rhythm of the heart's motions is not uncommon in persons advanced in life, in gouty subjects, and in individuals labouring under derangement of the digestive organs, accompanied by flatulence. It is frequently likewise met with in cases of disease of the valves, or of the muscular tissue of

\* Edinburgh Monthly Journal.

the heart, when it is often accompanied by irregularity, or inequality of the pulse, and it becomes then a sign of considerable importance.

By an *unequal* pulse we understand one in which some pulsations are strong and others weak. By an *irregular* pulse, one in which a few rapid beats are succeeded by one or more slower beats, and when the interval between them is different. Inequality and irregularity of the pulse are much more unfavourable signs than simple intermission, and are not observed except in cases of disease. Both are frequently met with in the same cases, and both accompany certain diseased states of the valves at the left side of the heart, of the muscular tissue of the organ, and of its investing membrane, the pericardium.

*Pulse in contraction of the left auriculo-ventricular Orifice.*—When the mitral valve or the left auriculo-ventricular orifice become diseased, so as to impede the passage of the blood from the left auricle into the left ventricle, the radial pulse will be weaker than natural, and will occasionally intermit. When this condition of the valve or orifice is more advanced, and the obstruction to the passage of the blood through the orifice becomes greater, the pulse, in addition to being weak and intermittent, will become small, irregular, and unequal, although the heart's action continues to be strong. Mr. Adams,\* of this city, was the first to call particular attention to the want of correspondence between the strength of the pulse at the wrist and the impulse of the heart in this form of disease: “the heart often beats so violently (he observes) as to shake the patient in his bed, while the pulse is small, weak, and irregular.” “I know not how,” he adds, “to describe it otherwise than by saying that it appears as if there were two pulses; one slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations.”

*Pulse in mitral regurgitation.*—When the mitral valve imperfectly closes the left auriculo-ventricular orifice, and regurgitation into the auricle occurs at each ventricular systole, the pulse will be scarcely affected if the aperture is very trifling; when larger, the pulse will become weak and small, and will intermit particularly when the circulation is hurried. When the mitral orifice is much enlarged, and permits a considerable portion of the

\* Dublin Hospital Reports, vol. iv.

3. In hypertrophy of the left ventricle, the pulse is "strong and prolonged." When dilatation is combined with the hypertrophy, the pulse is in addition full.

4. In dilatation of the left ventricle without hypertrophy, the pulse is large, but weak.

5. In dilatation with attenuation of the left ventricle, the pulse is weak and soft, easily quickened, and occasionally intermittent.

6. In patency of the aortic valves, the pulse is jerking, visible, and locomotive, but its frequency is not increased.

7. In diseased states of the coats of the arch of the aorta, with increase of the calibre of the vessel, the pulse has the same characters, but in a somewhat less marked degree.

8. In aneurism of the arch of the aorta, the pulse may have a different strength in each wrist, or it may be absent altogether in the radial artery on one side. It might also have a jerking character, resembling that of aortic regurgitation.

#### PULSE IRREGULAR.

1. In extreme contraction of the mitral orifice, the pulse is small, weak, intermittent, irregular, and unequal.

2. In regurgitation through the mitral orifice, when slight, the pulse is little altered; when extreme, the pulse has the same characters as in considerable contraction of the orifice.

3. In contraction of the aortic orifice, the pulse presents no peculiarity unless the degree of contraction is extreme, when it becomes small, irregular, and intermittent, rarely slower than natural.

4. In fatty degeneration of the muscular tissue of the left ventricle, the pulse, in the advanced stages, is small, weak, irregular, and unequal, sometimes slow.

5. In pericarditis with copious liquid effusion, the pulse presents somewhat similar characters.

6. In cases where fibrinous concretions form in the cavities of the heart, the pulse suddenly becomes small, weak, intermittent, and irregular.

7. In adhesion of the pericardium, the radial pulse is occasionally intermittent and unequal.

From what has been said, it may be gathered, that in several forms of cardiac disease, the characters presented by the pulse are

of a nature, materially, to aid the diagnosis : and if the different cardiac lesions were met with always distinct from each other, and uncomplicated with other affections, the signs derived from the pulse would be most valuable. Unfortunately, however, this is not often the case. Two or more forms of structural disease are frequently combined ; for instance, hypertrophy and dilatation of the left ventricle are very frequently associated ; and, as these morbid conditions are the ordinary result of disease of the valves, we may have obstructive or regurgitant disease of the aortic or mitral valves, or of both, combined with hypertrophy, or dilatation of the left ventricle, or with the two latter states. In addition, disease of any part of the heart may be associated with a state of anæmia or hysteria, with functional derangement, a gouty or a nervous habit, or with some of the other morbid conditions that have been alluded to ; when the pulse, if trusted to alone, would be more likely to lead into error, than to assist the diagnosis.

It must be borne in mind, too, that intermission or irregularity of the pulse may ensue in any diseased state of the heart, when the pulmonary circulation becomes greatly obstructed. It should be also recollected, that a very trifling morbid alteration of the valves at the left side of the heart, which may be accompanied by a loud *bruit de soufflet*, often causes no alteration of the pulse ; while, in a very advanced stage of the same disease, when the pulse is intermittent and irregular, an abnormal sound may be absent. Finally, as the systole of the right ventricle exercises no *direct* influence upon the pulse, the walls of the right side of the heart may be hypertrophied, or their cavities dilated ; or its valves or orifices might be diseased, without the pulse indicating in the slightest degree the morbid alteration.



## CHAPTER VIII.

SECONDARY SYMPTOMS OF CARDIAC DISEASE.—CONGESTION.—CONGESTION OF THE HEART, LUNGS, LIVER, INTESTINES, AND BRAIN.—POLYPOID CONCRETIONS IN THE CAVITIES OF THE HEART.—DROPSY.

THE secondary, the indirect, or the remote symptoms of cardiac disease, are, in the majority of cases, the result of some impediment to the return of the blood, conveyed by the pulmonary veins to the left side of the heart, or to that of the venous blood from the system generally through the right side of the organ, which in their turn are frequently the result of an obstruction to the free passage of the blood through the chambers of the heart. The blood, being retarded and delayed in the large veins, accumulates in the smaller vessels and capillaries; they become distended and dilated, their healthy state of elasticity or tone is impaired or diminished; and congestion is the result, which may be limited to the lungs and bronchial mucous membrane, or may extend to the liver, spleen, kidneys, and gastro-intestinal mucous membrane, causing more or less derangement of function in these organs.

When we speak of congestion, therefore, we mean a state in which there is a preternatural accumulation of blood in the minute veins and capillaries, with a weakened, retarded circulation, and a loss or deficiency of tone in the coats of the dilated vessels; in consequence of which they “react imperfectly upon the blood transmitted to them,” and are unable to “transmit the force of the current in the proper direction,” “vessels which have lost their tone becoming inelastic and tortuous, and, by the very stagnancy of the blood in them, opposing an increasing obstacle to its passage through them.”

The cause of the motion of the blood in the capillaries, is now generally considered to lie in the impulse given to this fluid by the contraction of the ventricle. An experiment which was performed by Dr. Sharpey seems to confirm this. “A syringe, with



a hæmadynamometer, to show the amount of pressure used, was adapted to the aorta of a recently dead animal, the vena cava being divided; warm water was then injected, and, with a force which raised the mercury in the hæmadynamometer only three inches, the water passed through the capillaries, and out of the vena cava. When the pressure was increased so as to raise the mercury six inches, the flow was very free; and on adapting another hæmadynamometer to the vein, the pressure in this, was found to rise as high as three inches. The pressure thus used in the arteries (six inches of mercury) was not greater than the natural pressure in the arteries of a living animal; and the pressure transmitted to the veins (three inches of mercury) was greater than that in the veins of a living animal—thus, showing, that the force of the heart, sustained by arterial tension, is quite adequate to effect the circulation without other aid.” Hence, when the contractile power of the left ventricle is weakened, owing to dilatation of its cavity, to attenuation of its parietes, or to softening of its tissue, the blood must necessarily pass with greater difficulty through the systemic capillaries; while, if the circulation through the left side of the heart is impeded, the capillaries of the lungs will become congested in the first instance, followed by general venous congestion and the train of symptoms presently to be described.

It was at one time a very general opinion, that cerebral congestion and apoplexy on the one hand, or hæmoptysis and pulmonary apoplexy on the other, had their cause, the former in hypertrophy of the left ventricle, the latter in hypertrophy of the right; their occurrence was, in fact, looked upon simply as an example of cause and effect, the increased force with which the hypertrophied ventricle transmitted the blood to the brain or lungs being considered sufficient to produce congestion in these organs, terminating in serous or sanguineous effusion, or in rupture of the coats of the small vessels. This theory is, however, ill supported by pathological facts and, so far as the lungs are concerned, may be said to be exploded; the congestion which occurs in the latter organs, as the result of cardiac disease, being much more frequently the result of obstruction to the free passage of the blood through the chambers of the left side of the heart. Because, even though the propulsive power of the ventricles is increased, if there is no impediment to the return of the blood by

the veins, the circulation will only be carried on more vigorously ; the veins returning the blood as rapidly as it is transmitted by the arteries. Indeed, congestion is much more liable to occur when the force with which the blood is propelled by the ventricles is diminished, as in dilatation and attenuation of the ventricles or in softening of the tissue of the heart, than when this fluid is propelled with increased force by an hypertrophied ventricle.

Dr. Wardrop\* has recently called attention to a function performed by the lungs, and to another by the subcutaneous veins, by which, he is of opinion, congestion is prevented ; the former he terms the *pulmo-cardiac* function ; the latter, the *veno-pulmonary* function.

*Pulmo-cardiac function.*—“ Whenever the systemic blood cannot find a ready exit from the left ventricle, and when, at the same moment, there is no diminution in the supply of venous blood to the right auricle, an accumulation or congestion of blood (Dr. Wardrop observes) must take place within the cavities of the heart ; and, therefore, in order to prevent such undue accumulation, the effects of which would be more or less injurious” an accessory function of the lungs comes into operation ; “ the pulmonary vessels serving as a *receptaculum*, or reservoir, for receiving any surplus quantity of blood, whether venous or arterial, which the cavities of the heart cannot admit.”

“ To fulfil this *pulmo-cardiac* function, the structure of the lungs is admirably adapted. The pulmonary vessels, being imbedded in a soft and yielding substance, are susceptible of various degrees of distention, so that they readily give way for the reception of any surplus quantity, whether of venous or of arterial blood, and retain it until it can be received within the heart. The very same structure with which the lungs are endowed to enable the air-cells to accommodate themselves to those differences in the quantity of air which take place during respiration, also enables the pulmonary vessels, in their turn, to accommodate themselves for the reception of the various quantities of blood which may be impelled into them.”

“ A function is, therefore, performed by the respiratory organ which is quite unconnected either with arterializing the blood with aiding the return of the venous blood to the right heart

\* On Diseases of the Heart.

or with assisting the circulation in the pulmonary vessels; and when it is considered how often, and by what slight causes, the current of the arterial blood is hindered from leaving the left heart; and, how frequently the return of the venous blood is accelerated to the right heart, the *pulmo-cardiac* function must be regarded as one of primary importance."

*Veno-pulmonary function.*—While the pulmo-cardiac function is employed to relieve the heart of any surplus quantity of blood which it cannot receive within its cavities, the *veins* will, in like manner (Dr. Wardrop observes) be found to relieve the pulmonary vessels of any superabundant blood which they are not capable of receiving without interruption to the great function of respiration. This office of the veins, he terms the *veno-pulmonary* function. "In the limbs, the deep-seated veins which are intermingled with the muscles are influenced by every muscular contraction; whereas the subcutaneous or superficial veins, being placed external to the *fascia*, are not affected by the action of the muscles of the extremities. Whilst, therefore, the chief function of the veins is to convey the blood back to the heart, there are likewise other offices especially executed by each of these subdivisions of the venous system."

"No more striking example can be given of this office of the subcutaneous veins, than their distended appearance in the limbs of a person after making any violent exertion, or beneath the delicate skin of a race-horse after a severe gallop." "But neither this accessory function of the subcutaneous veins, the increase in the heart's action, nor the aid which the respiratory organs afford, are always (he observes) sufficient to prevent a congestion of blood within the cavity of the chest."

*Effects of congestion.*—When congestion is carried to an extreme degree, or has lasted for a considerable time, the overloaded or overdistended vessels relieve themselves by allowing the more watery parts of the blood to transude, and serous effusion occurs, or the blood itself may escape, or rupture of a vessel and extravasation of blood may occur. Long-continued congestion frequently leads to more or less alteration of the tissue of the congested organ; its size is frequently increased; and this may or may not be accompanied by pain, or by tenderness on pressure. This is most frequently witnessed in the liver, and, if the cause

continues long in operation, the increase in size may be permanent. Dr. Clendinning\* has shown that the lungs acquire increased development and weight under such circumstances; "the inter-vesicular and interlobular textures becoming, Dr. Williams observes, hypertrophied, with partial consolidation of the vesicular structure itself." These changes are best marked in the posterior part of the lungs, near their roots.

In the great majority of cases, congestion is owing either to—  
1st. Feebleness of the heart's action, the result of dilatation of the left ventricle, of attenuation of its parietes, or of softening of its muscular tissue, or to two of them combined; in consequence of which the contractile power of the left ventricle is diminished, and its systole is insufficient to propel the blood through the systemic capillaries; 2ndly. To free regurgitation through the tricuspid orifice, the result of dilatation of this opening, combined with hypertrophy and dilatation of the right ventricle; by which the return of the blood from the *venæ cavæ* to the right auricle is greatly impeded, leading to its accumulation, first in the large venous trunks, and next in the smaller veins and capillaries; and 3rdly. To a contracted state of the mitral orifice, impeding the passage of the blood from the left auricle into the left ventricle, or to a state of the mitral valve or orifice which permits free regurgitation through it; owing to which much of the contents of the left ventricle is transmitted backwards into the left auricle at each systole. In the first of these cases the congestion commences in the capillaries of the systemic circulation, and extends to the veins; in the second it commences in the large veins, and extends to the capillaries; in the third it commences in the pulmonary capillaries and extends to the large veins.

In addition, it has been shown by MM. Becquerel and Rodier,† that the effect of long-standing disease of the heart is to occasion changes in the quality and relative constituents of the blood. Thus, the globules undergo diminution in advanced cases of disease of this organ; the fibrin is likewise diminished remarkably, manifested by the occurrence of hæmorrhage from mucous surfaces. The albumen of the serum of the blood is also diminished, by which the occurrence of dropsy is favoured; and, there is in addition an increase in the watery part of the blood.

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In the slighter forms of congestion, the functions of the organ the seat of the congestion may be little affected: in the advanced stage its functions are frequently seriously impaired. Thus in congestion of the lungs and bronchial mucous membrane we have dyspnoea, passing into orthopnoea, with oppression, cough, and expectoration, which may or may not be followed by hæmoptysis or pulmonary apoplexy; the secretion from the bronchial mucous membrane is often remarkably increased, and this may prove one of the most troublesome symptoms. Congestion of the liver is accompanied by an increase in size of the organ, and by impeded or vitiated secretion, or by arrest of the biliary secretion, frequently with pain or tenderness on pressure. When the gastric mucous membrane is congested, the disturbance of function may be indicated by increased or altered secretion, or we may have nausea, or antiperistaltic action of the organ, or pain may be the most prominent symptom, or loss of appetite may be alone complained of. When the spleen is congested, the organ sometimes attains a remarkable size; this, however, is more frequently observed as an effect of ague than of cardiac disease. When the liver and gastro-intestinal mucous membrane are both congested, we may have pain, deranged digestion, loss of appetite, flatulent distension, vomiting, hepatic derangement, hæmatemesis, diarrhoea, hæmorrhoids, jaundice, or ascites. When the brain is congested, its sensibility is sometimes impaired, sometimes increased, and we may have headache, tinnitus aurium, vertigo, sense of weight in the head, confused vision, or disordered volition, or a semi-comatose state, which, in extreme cases, may terminate fatally, with or without the rupture of any vessel. Finally, when the kidneys are congested, the urine becomes scanty, high-coloured, turbid, and albuminous, followed by anasarca which is slight at first, and commences in the most dependent parts, but eventually becomes general.

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## CONGESTION OF THE HEART.

The circulation through the chambers of the heart cannot be impeded without the coronary circulation suffering; and this circulation cannot be deranged without the functions of the heart being disturbed.

The integrity of a muscle, and its healthy action depend in a great measure, we know, upon its receiving a due supply of arterial blood; if this is sufficient, and of a good quality, the functions of the muscle will be performed with vigour; while if the supply is insufficient, or the quality is deteriorated, the functions of the muscle will be impaired. Now, the heart being a muscular viscus, requires for the vigorous performance of its functions an efficient supply of arterialized blood; and although the blood which circulates through its chambers is the ordinary stimulus to the motions of the organ, if the coronary vessels become distended and overloaded, and the coronary veins are unable to empty themselves; or, if the coronary arteries convey blood of deteriorated quality, congestion of its tissue must ensue, and its functions must be imperfectly performed.

Thus when the circulation through the chambers of the heart has been long impeded, when considerable obstruction exists in the pulmonary circulation, and the venous blood does not undergo the necessary changes in the lungs, the coronary circulation becomes not only impeded and retarded, but the coronary arteries convey a mixture of venous and arterial blood to the tissue of the heart. The functions of the muscle are in consequence impaired; its irritability diminishes, it is no longer obedient to the stimulus of the blood which distends its cavities, and its action becomes feeble and intermittent, or unequal and irregular. Indeed, the immediate cause of death in valvular and other disease of the heart is probably often the result of impeded coronary circulation.

## CONGESTION OF THE LUNGS.

The lungs, from their physiological position between the right and left chambers of the heart, must suffer whenever the circulation through the heart is not free, or when disease to any extent exists in the valves or orifices of the left side of the organ. Thus when the mitral orifice is contracted, the left auricle, being

unable to empty itself, becomes distended, and the blood returning by the pulmonary veins cannot freely enter the auricle; the right ventricle at the same time continuing to transmit the blood by the pulmonary artery, the capillaries of the lungs become overloaded, and the pulmonary tissue and the bronchial mucous membrane congested. When the mitral valve imperfectly fulfils its functions, and much of the contents of the ventricle is transmitted backwards at each systole, the same results may follow as where the mitral orifice is contracted. When the aortic orifice is diseased, so as to obstruct the orifice considerably, the same results sometimes follow; but as Dr. Blakiston observes, a considerable amount of disease of the aortic orifice may exist for a number of years without seriously affecting the general health, more particularly if hypertrophy of the left ventricle is joined with it.

The congestion of the lungs and bronchial mucous membrane which occurs under such circumstances is always of the passive form, and is always a secondary or consecutive lesion. When it has lasted long, or when the patient has suffered frequent attacks of it, a condition of the part remains, in which, inflammation or a state approaching to it, is very liable to be set up, and sometimes by causes which would be incapable of giving rise to it in a healthy condition of the parts: hence we not unfrequently observe in addition a real determination of blood to the lungs, the result of inflammation of the parenchyma of these organs.

The important function which the lungs perform in the conversion of venous into arterial blood must obviously be seriously interfered with when the circulation through the pulmonary capillaries is retarded and impeded, and the minute vessels are considerably engorged. The capillary vessels of the lungs form, we know, a net-work around each vesicle or air-cell; they are exceedingly numerous, and in greater abundance in a given space here than in almost any other part of the body. Now as the blood must all pass through them before it arrives at the commencement of the systemic circulation, it is obvious that any impediment to its passage will be felt throughout the system; while, if the blood is prevented from undergoing the change from venous to arterial in the pulmonary capillaries, the pulmonary veins will convey venous as well as arterial blood to the left auricle. Hence, when the congestion is considerable in degree, and engages

much of the pulmonary tissue, and the air-cells of the part scarcely permit of the entrance of atmospheric air during inspiration, the blood will be imperfectly oxygenated, its carbonic acid will be incompletely removed, and the renewal of the fibrine will be imperfectly accomplished; a mixture of venous and arterial blood will then circulate in the arteries, which is unfit for maintaining the functions of the various organs in a healthy condition, or for supplying nutriment to their tissue; the brain, the nervous system, the heart, and the other organs, the maintenance of the function of which depends upon a due supply of arterial blood, suffer; the muscular system receives less fibrine, the animal heat diminishes, and the entire system feels the depressing influence.

When the pulmonary capillaries have been congested for a long time, or to a considerable degree, the overloaded vessels are relieved by the transudation of the watery, saline, albuminous, and sometimes of the fibrinous constituents of the blood; or the blood itself may be effused upon the surface of the bronchial mucous membrane, or into the air-cells; or rupture of a vessel may take place, with extravasation of blood upon the mucous surface, or into the pulmonary tissue; accompanied by œdema of the lungs, dyspnœa (which in extreme cases amounts to orthopnœa), cough, and watery expectoration; or hæmoptysis, and pulmonary apoplexy.

Long-continued congestion not only interferes with the functions of the congested organ, but it also frequently affects its nutrition and structure. "It generally tends" as Dr. Williams observes "to cause an increased deposit in them, constituting a variety of hypertrophy." "In the lungs, long-continued congestion may" he remarks, "cause hypertrophy of the intervesicular and interlobular textures, and in some cases partial consolidation of the vesicular structure itself." "Such changes" he adds "are frequently met with in connexion with long-standing disease of the heart, and abound most in the posterior parts of the lungs and near their roots, the most vascular parts."

*Œdema of the Lungs.*—When the congested capillaries of the lungs have relieved themselves by allowing the serous or thinnest portion of the blood to transude; and when the air-cells, the minute ramifications of the bronchial tubes, and the interlobular cellular tissue have become infiltrated with this fluid, it constitutes

the state known under the name of œdema of the lungs; and, as in œdema, or anasarca of the extremities, the fluid gravitates to the most dependent parts, and will generally be found to occupy the base of the lungs, or the posterior parts of these organs.

Œdema of the pulmonary tissue is necessarily therefore a symptom only in the advanced stages of cardiac disease, and the forms of disease with which it is usually associated are those in which a considerable impediment exists to the passage of the blood through the left chambers of the heart, particularly valvular disease. It is most common in the obstructive lesion of the mitral orifice; indeed, few cases of this form of disease go through their stages without this condition being present in a greater or less degree. It is usually preceded or followed by anasarca of the cellular tissue, and is always accompanied by more or less dyspnœa, cough, and expectoration, frequently of a thin mucous or serous fluid, as well as by certain physical signs, the most remarkable of which are dullness on percussion, and a moist sub-crepitant râle on auscultation of the base of the lungs.

Œdema of the lungs, though no uncommon effect of cardiac disease, is by no means limited to diseased states of the heart, but is met with in several other morbid conditions; thus it may accompany general dropsy, it is observed in chronic bronchitis, and pertussis; it follows the resolution of pneumonia; and it is met with as a sequela of measles and scarlatina.

*Dyspnœa.*—The respiratory movements, like the motions of the heart, are involuntary, though the former are much more under the influence of the will than the latter: they belong to the class of movements termed “excito-motory” by physiologists. The object of the respiratory process, it is scarcely necessary to say, is to bring the venous blood in contact with atmospheric air, which is accomplished through the medium of the innumerable capillaries that ramify upon the air-cells of the lungs; and the changes which ensue in the blood and in the air take place through the delicate membranous walls of these cells. From the manner in which these parts are arranged, every particle of the blood, in its passage through the capillaries of the lungs, is exposed to the action of the oxygen of the air; and anything which impedes or interrupts the process tends to derange or to disturb the general circulation.

When the respiration is perfectly tranquil, the respiratory movements are performed almost entirely by the diaphragm, which, contracting in inspiration, enlarges the diameter of the thorax from above downwards, and relaxing in expiration, returns to its former state. When the respiratory movements are carried on more actively, the thorax is enlarged in all its diameters—the antero-posterior, lateral, and vertical. When, finally, the respiration becomes difficult and laborious, all the muscles, in addition, which receive filaments from the system of respiratory nerves are brought into action.

In a state of health, the exact amount of venous blood is transmitted by the right ventricle to the pulmonary artery which can be converted into arterial blood in the lungs; and the exact amount of arterial blood is returned, by the pulmonary veins to the left auricle, which will pass freely through the chambers of the left side of the organ. At the same time, an amount of atmospheric air is taken into the lungs at each inspiration, sufficient to convert the blood contained in its capillaries into arterial blood. We have likewise seen that, in a state of health, there is an exact ratio between the pulsations of the heart and the respirations in a given period: thus, when the circulation is hurried by exercise, the respiration becomes more frequent in proportion; and, as the circulation becomes again tranquil, the respirations diminish in frequency. There is, therefore, in health, a perfect equilibrium between the circulation and the respiration, and if from any cause this equilibrium is disturbed, dyspnœa ensues.

It sometimes happens that the blood is transmitted with too little force, by the right ventricle, to pass freely through the pulmonary capillaries: more frequently, however, the blood is impeded in the latter vessels, owing to some obstruction to its passage through the left chambers of the heart. Again, it sometimes happens that the blood is transmitted with increased force, or in greater quantity than natural, by the right ventricle: more frequently, however, the air cannot enter the air-cells of the lungs in sufficient quantity to decarbonize the blood transmitted to these organs. In either case, there will be a want of due proportion between the extent of the oxygenating surface and the amount of blood; the respiration will become hurried, or difficult, or laborious, according to circumstances; the dyspnœa

will pass into orthopnœa, which, in extreme cases, may terminate in asphyxia.

Dyspnœa will therefore ensue whenever the pulmonary capillaries contain more blood than can be oxygenated by the air admitted into the air-cells, or whenever too little air can enter the air-cells to oxygenate the blood contained in the pulmonary capillaries. In either case, the respirations may be more frequent than natural, or the number of the respirations and of the pulsations of the heart may not bear their normal proportion to one another. Under such circumstances, the respiration is no longer an involuntary act, but requires the aid of a number of the voluntary muscles, particularly in inspiration, in order that a sufficiency of air may be taken in to convert the venous into arterial blood. In extreme cases all the muscles which receive branches from the respiratory nerves—viz., the facial, the external respiratory nerve, the spinal accessory, the glosso-pharyngeal, the par-vagus and its laryngeal branches, and the spinal nerves distributed to the muscles of the trunk—are called into almost convulsive action.

Dyspnœa, in its mildest form, when the individual is merely short-winded, or readily put out of breath, or finds a difficulty in ascending stairs, although a frequent attendant upon disease of the heart, is observed in many cases where this organ is not in the least engaged. Indeed, dyspnœa is seldom marked in cardiac disease until the pulmonary tissue and the bronchial mucous membrane become secondarily engaged, and it is then often one of the most distressing symptoms. Dyspnœa, to a considerable extent, may however exist, and yet the patient will deny its existence, either unconscious of it or regarding it as too trifling to be mentioned. This is not unfrequently witnessed in the class of patients whom we see in hospital, in whom the dyspnœa has made its approaches so gradually that they have become habituated to it. It, no doubt, is in some measure due to the parts having had time to adapt themselves to the altered circumstances, and in some measure, also, “to the sensibility being gradually blunted by the circulation of ill-oxygenated blood.” Thus, as Dr. Williams\* observes, “Persons affected with extensive emphysema of the lungs are habituated to an imperfect state of respiration, which is shown by a constant lividity of the lips and cheeks: such an

\* Principles of Medicine.



appearance would be a sign of approaching death in other persons."

The severity of the dyspnœa, both in disease of the heart and lungs, is, in the majority of cases, in proportion to the suddenness with which this symptom has set in: thus in capillary bronchitis, in pneumonia engaging a considerable portion of both lungs, in pleuritis with copious effusion, it is the most prominent symptom. It is likewise so in the acute forms of cardiac disease, as where a large amount of fluid is suddenly effused into the pericardial sac—the result of pericarditis; or where the circulation through the heart becomes suddenly impeded or obstructed; as occurs in some of those cases of acute endocarditis which prove rapidly fatal.

When more or less dyspnœa is constantly present, and yet no signs of pulmonary disease can be detected, or there is no manifest cause for its presence, "we may," Dr. Furnivall\* observes, "suspect disease of the heart almost from this symptom alone." In some cases which he met, "this was the only symptom for a long time observable, and it appeared long before the characteristic murmur of the diseased mitral valve could be detected."

The diseased states of the heart, in which dyspnœa is a prominent symptom, are—pericarditis, with copious liquid effusion; endocarditis, with obstruction in the orifices or chambers of the heart; dilatation of the ventricles, with thinning of the parietes; hypertrophy of the ventricles in an advanced stage, and a combination of hypertrophy with dilatation; valvular disease, particularly considerable contraction of the orifices of the left side of the heart, or free regurgitation through the mitral orifice; or aneurism of the aorta compressing the trachea, or large bronchi. Dyspnœa, however, is not limited to cases of organic disease of the heart; it is a frequent symptom of the anæmic, or of the plethoric state. In such cases, however, the dyspnœa is not constant, but is experienced on walking quickly, or ascending stairs, or on making any unusual exertion. In the former of these cases the dyspnœa, according to Andral, arises from there being too much air relatively to the quantity of blood to be oxygenated; in the latter, from there being too much blood relatively to the air entering the bronchial tubes.

Patients labouring under valvular disease, in an aggravated

\* The Lancet, 1846.



form, complicated with dilatation of the ventricles, and accompanied by congestion of the pulmonary tissue, are predisposed to attacks of bronchitis or pneumonia, which aggravate remarkably the habitual dyspnoea, and usually hurry on the disease to a fatal termination.

*Starting in alarm from sleep.*—In connexion with dyspnoea, a symptom sometimes observed in cases of heart-disease of long standing is a sudden starting in alarm from sleep, accompanied by a distressing feeling of oppression and violent action of the heart, often following a frightful dream. This symptom, Dr. Willis\* observes, is probably connected with impeded circulation through the lungs. “As we fall asleep,” he observes, “the respiration becomes considerably slower than it was immediately before: in very deep sleep each respiration is an effort, and is apparently only performed from the increasing urgency of the uneasy sensation that is at once allayed by taking in a new draught of air. The pulse at the same time falls in frequency, and the balance is maintained between the activity of the circulation and that of the respiration. But, with a heart acting faultily, pushing rather more blood, perchance, into the pulmonary artery than it can transmit, or, on the contrary, refusing readily to receive so much as returns to it by the pulmonary veins, the balance between the activity of the circulation and that of the respiratory system is destroyed, accumulation takes place in the lungs, the patient awakens in alarm, and, plying the muscles of respiration more vigorously, shakes off the sense of suffocation that was beginning to be imminent.”

*Cough.*—Cough is not a necessary symptom of cardiac disease; few cases, however, go through all their stages without this symptom being present in a more or less marked degree, particularly if congestion of the lungs ensues. The act of coughing, which consists in a sudden voluntary or involuntary expiratory effort, is almost always the result of some irritation of the mucous membrane lining the larynx, trachea, or its ramifications. The nature of this irritation varies in different cases; thus it may depend upon inflammation of some portion of the mucous membrane which lines the air-passages, in consequence of which the sensibility of the membrane is increased, and its secretions, which are not irri-

\* London Medical Gazette.

tative in a healthy state of the membrane, become so, and occasion cough; or it may arise from the accumulation of the secretion in the air-tubes, by which the free admission of air is interfered with, and cough is excited in order to get rid of it. Again, it sometimes happens that the calibre of the air-tubes is diminished by submucous infiltration, or by a morbid growth pressing upon and flattening one, when the irritation excited gives rise to cough.

When cough arises in diseased states of the heart, it may depend upon any of the causes just mentioned; most frequently, however, it arises from the irritation occasioned by the increased quantity of fluid poured out into the air-passages, the amount of which is sometimes considerable, and which, by its simple presence, excites irritation, or, by its quantity or situation, impedes the passage of the air during respiration, and cough is excited in order to get rid of it.

In the early stages of cardiac disease, if cough is present, it is in general due to an accompanying bronchitis. In the advanced stage of certain forms of disease of the organ, cough is scarcely ever absent, and it is always associated with more or less dyspnoea. In such cases, oedema of the pulmonary tissue is generally present; the congested condition of the lungs being in part relieved by the transudation of the more watery parts of the blood into the air-cells and minute tubes: but this very effort of nature to relieve the congested state of the capillaries of the lungs becomes often the source of considerable distress to the patient; the copious secretion poured out, as it can only be removed by coughing, excites and keeps this up; while, if the secretion from the bronchial mucous membrane is at the same time very profuse, and the patient much debilitated, the expectoration becomes difficult, and not unfrequently the patient dies asphyxiated from this very cause.

The diseased states of the heart in which cough is most frequently a symptom, are the same as those in which dyspnoea is observed; indeed, the latter is seldom prominently marked, without cough being likewise troublesome. The cough at first may be dry, or accompanied by expectoration; when congestion or oedema of the lungs supervenes, it becomes free, the expectoration is copious, and consists frequently of a colourless, watery, or muco-serous fluid. The cough, too, comes on in paroxysms, and in the advanced stage the fits are sometimes both frequent and prolonged.

by which congestion of the lungs is still further increased. Patients in whom the pulmonary circulation is much obstructed, and in whom congestion of the lungs has lasted long, appear likewise to be more subject than others to bronchitis, by which the habitual dyspnoea and cough are considerably aggravated.

It is scarcely necessary to observe, that whenever the trachea, or a large bronchial tube, is compressed by the growth of an aneurismal or other tumor in the cavity of the thorax, cough is a prominent symptom. In such cases, the sensation experienced by the patient is, often, as if something existed in the air-tubes which he would be relieved by expectorating, and he makes many ineffectual efforts to do so. Eventually, the act of inspiration or of expiration, or both, are accompanied by peculiar sounds, which in general are sufficiently characteristic, and when once heard can scarcely be mistaken.

*Hæmoptysis.*—The blood which escapes upon the mucous surface of the air passages, and is expectorated, may have its source either in the *bronchial* or the *pulmonary* vessels; and may come, either from a ruptured vessel or it may transude without the rupture of any vessel. We have already seen how intimately related the functions of the heart are to those of the lungs; and how liable the pulmonary circulation is to be impeded when the valves or orifices of the heart are diseased, and prevent the free passage of the blood through its chambers. We have likewise seen the manner in which the over-distended capillaries of the lungs are relieved by the transudation of the serous portion of the blood, giving rise to œdema of the pulmonary tissue. If the impediment to the passage of the blood becomes still more considerable, the capillaries may relieve themselves by allowing the blood itself to transude, when it will be expectorated; or rupture of a vessel may take place, and the blood be effused into the tissue of the lungs, constituting the state termed pulmonary apoplexy.

Hæmoptysis is a symptom only of the advanced stage of cardiac disease, and is limited to certain forms of it; it is most common in young subjects, and in individuals who at some former period had laboured under acute endocarditis. It was at one time a common opinion, and is probably still with some, that when hæmoptysis takes place in the progress of cardiac disease, or when pulmonary apoplexy occurs in such cases, they depend upon

hypertrophy of the right ventricle, transmitting the blood to the lungs with so much increased force as to cause rupture of the minute vessels, and effusion of blood, either upon the mucous surface or into the pulmonary tissue. This, however, is not correct view of the subject; because, even though hypertrophy of the right ventricle did exist, the blood would find no difficulty in returning by the pulmonary veins to the left side of the heart. But hypertrophy of the right ventricle is itself almost always secondary lesion, often of valvular disease at the left side; particularly contraction of the mitral orifice. We have seen that this form of valvular disease is a frequent cause of pulmonary congestion; and, if hypertrophy of the right side is superadded, the pulmonary circulation being placed between a strong right ventricle propelling the blood with increased force into the pulmonary artery, and a contracted state of the left auriculo-ventricular orifice, by which the pulmonary veins are prevented from freely pouring their blood into the left auricle, it is not surprising that the blood-vessels should relieve themselves by allowing the contents to transude, or that rupture of a vessel should take place. Hæmoptysis would be much more frequent in such cases, if, as the right ventricle became hypertrophied and dilated, the tricuspid orifice preserved its normal dimensions; but as a general rule almost, when the cavity of the right ventricle becomes dilated and its walls hypertrophied, the tricuspid orifice increases in size by which free regurgitation is permitted into the right auricle, part of the strength of the ventricular systole is thus wasted, and the injurious consequences which might otherwise ensue are prevented.

Hæmoptysis is a more common symptom of pulmonary than of cardiac disease; the source of the hæmorrhage is not the same, however, in the two cases: the appearance of the blood is likewise often somewhat dissimilar, while the circumstances under which the hæmorrhage occurs and the condition of the circulation at the time, are different. For instance, the hæmoptysis which occurs in phthisis is of the active form, and arises from determination of blood; the general circulation is quickened, and the hæmorrhage comes (as was first pointed out by Dr. Graves\*) from the *bronchial vessels*. Pure blood is frequently expectorated, which

\* Clinical Lectures.

frothy, and of a bright florid hue, though it is often also in clots, and of a dark colour if it has lain for any time in the air-tubes: the quantity expectorated is sometimes considerable, and in the intervals the sputa are frequently streaked with blood: finally, this form of hæmoptysis is seldom associated with pulmonary apoplexy. On the other hand, the hæmoptysis which occurs in the advanced stage of cardiac disease is of the passive form, and is the result of venous congestion not of local determination, while the circulation is not quickened generally. The blood expectorated comes from the *pulmonary vessels*, and the amount is sometimes considerable. This will, however, in some measure, depend upon whether the hæmorrhage is owing to rupture of a vessel, or depends simply upon the transudation of the blood: in the former case the blood may have a florid hue, or it may present a dark or grumous appearance; in the latter, the blood is frequently intimately mixed with the sputa. Finally, hæmoptysis from this cause is occasionally associated with pulmonary apoplexy.

The diseased conditions of the heart in which hæmoptysis most frequently occurs are :

- 1st. Considerable contraction of the mitral orifice.
- 2nd. Dilatation with attenuation of the left ventricle.
- 3rd. Extreme contraction of the aortic orifice.
- 4th. Hypertrophy and dilatation of the right ventricle. This, however, is very often consecutive to valvular disease of the left side of the heart.

Few cases of considerable contraction of the left auriculo-ventricular orifice go through all their stages without the occurrence of some hæmoptysis. It is rare in the regurgitant lesions of the orifices of the left side of the heart; particularly so in aortic regurgitation.

The explanation of the cause of the hæmoptysis in all these cases is pretty nearly the same. Thus, when the mitral orifice is contracted, and the passage of the blood through it is impeded, congestion of the lungs in general sooner or later follows; particularly if the parietes of the right ventricle become hypertrophied: under such circumstances, any sudden or continued exertion on the part of the patient, or anything which hurries the heart's action, may give rise to hæmoptysis, the over-distended vessels allowing of the transudation of the blood, or rupture of a vessel ensuing. When,

again, the cavity of the left ventricle is dilated, and its parietes are attenuated, the weakened ventricle is unable to propel its contents with sufficient force to pass through the capillaries; the blood accumulates in the left cavities of the heart, the pulmonary veins are unable to empty themselves, congestion of the lung ensues, followed as in the preceding case by hæmoptysis.

*Pulmonary apoplexy.*—When the blood, in addition to being expectorated, is extravasated in the tissue of the lung, it constitutes the state with which we are familiar as pulmonary apoplexy. There is then always rupture of a vessel, and the effused blood may fill the air-cells, or the areolar tissue of the lungs may be infiltrated with this fluid, by which the air-cells of the part are compressed or obliterated, and no longer permit of the entrance of air.

Pulmonary apoplexy, although very generally accompanied by hæmoptysis, may occur without it; and, according to its extent, to the quantity of blood extravasated, and to the amount of lung engaged, it may prove immediately fatal, or only so after a longer or shorter interval; or, the patient may recover. The attack may be sudden or otherwise, and the symptoms will vary in intensity according to the circumstances just mentioned. It is usually accompanied by increase of palpitation and dyspnoea, by oppression of the chest, by pain referred to some part of the front of the chest, or to the sides, or shoulders, or by a sensation of heat in the chest—by anxiety, by cough with more or less hæmoptysis, by paleness of the face if the amount of blood expectorated is considerable, or by injection of the face if it is less, as well as by certain physical signs upon which it is not necessary to dwell here.

The diseased states of the heart in which pulmonary apoplexy is most likely to supervene are those in which the impediment to the return of the blood to the left side of the heart is greatest: consequently, it is most frequently observed in diseased states of the left auriculo-ventricular orifice; particularly in a very contracted state of this orifice. When this morbid condition has followed rapidly upon an attack of acute endocarditis, and when the subject is still young, pulmonary apoplexy, as M. Gendrin\* observes, is more liable to occur than when the disease has come

\* Leçons sur les Maladies du Cœur.

on gradually, and when the congested state of the lungs has been in some measure relieved by the escape of the serous portion of the blood, constituting œdema of the lung, or when anasarca of the cellular tissue has supervened.

## CONGESTION OF THE LIVER.

The connexion which exists between a congested state of the liver and morbid conditions of the heart, was alluded to by Corvisart, and has been noticed more or less by succeeding writers. Indeed, next to the lungs, the liver is more liable than any other organ to become congested, as the result of cardiac disease; and there are several good anatomical reasons why this should be so. 1st. Owing to the structure of the liver; 2nd. To the large amount of blood which circulates through it; 3rd. To the blood which reaches it by the vena portæ having already passed through capillary vessels, and having consequently lost whatever influence the contraction of the left ventricle had upon it; 4th. To this blood having to pass through two sets of venous capillaries before it reaches the branches of the venæ cavæ hepaticæ; and 5th. To the returning blood of the liver having to arrive at the right side of the heart against gravity.

The substance of the liver, in general terms, may be said to consist of a close net-work of capillary vessels and ducts, arranged in lobules, and enclosing in their meshes nucleated cells; the tissue of the organ is soft and yielding, and it readily becomes increased in size when its vessels are distended. Again, the whole of the venous blood from the abdominal viscera (with the exception of that from the kidneys, bladder, and uterus) must pass through the liver before it can reach the right side of the heart; which large amount of blood circulates through the capillaries of the liver, in addition to that which it receives by its own nutritious artery. The circulation of this venous fluid through the liver must necessarily be slow, in consequence not only of the absence of the influence of the contraction of the left ventricle, but from the large size of the capillaries, and the blood having, as has already been observed, to pass through two sets of venous capillaries. Now, if any impediment exists to the passage of the blood through the right chambers of the heart, owing to disease seated in the lungs, or in the left side of the heart: or if the blood as-



ascending by the inferior cava cannot freely enter the right auricle, the circulation in the *venæ cavæ hepaticæ* becomes retarded, which reacts through the intermediate vessels upon its capillaries. The capillaries of the *venæ cavæ hepaticæ* have their origin in the centre of each of the lobules, and the circulation through them being impeded, they, as well as the intermediate vessels, become distended. This constitutes the slightest form of congestion of the liver, and has been termed by Mr. Kiernan “the first stage of *hepatic venous congestion*.” A section of the liver under such circumstances presents a mottled appearance, the centre of each lobule having a deeper colour than the rest. When the impediment to the return of the blood to the right side of the heart increases, the capillaries of the *venæ portæ*, which occupy the circumference of the lobules, next suffer. The blood in both sets of capillaries being retarded, they become distended, constituting a still more advanced stage of congestion of the liver; or, what Mr. Kiernan calls “the second stage of *hepatic venous congestion*.” Here “a section of the liver presents likewise a mottled appearance, but now the pale portion will be in spots.”

Both sets of vessels may be congested without the function of the liver being impaired, or without any considerable impediment to the secretion or escape of the bile. In many cases, however, when the congestion has lasted long, and the impediment to the return of the blood to the right side of the heart is considerable, the biliary plexus, and the minute ducts which ultimately form the excretory duct, suffer from the pressure; the bile is prevented from freely escaping, it accumulates in the lobules, and biliary is superadded to sanguineous congestion. A section of a portion of the liver will then present the appearance with which we are familiar as *nutmeg liver*.

Congestion of the liver may, therefore, be limited to the ramifications of the *venæ cavæ hepaticæ*, or it may extend to the capillaries of the *venæ portæ*; or both may be combined with biliary congestion. The first has been termed the passive form of congestion, the second the active form; but, when congestion of the liver depends upon cardiac disease, it is always of the passive form. The congestion of the liver may be partial, and occupy only a portion of the organ, or it may engage the entire viscus, and this cannot occur without occasioning an increase in the size



of the organ, which will be in proportion to the degree of the congestion; to the increased amount of blood contained in the distended vessels; to the length of time the causes which produced it have been in operation; and to the condition of the liver at the time. "In young subjects, and in persons in whom the liver is healthy, and its capsule thin, Dr. Budd\* observes, the liver will necessarily enlarge much more for a given force of distension than in persons in opposite circumstances." "When the liver (he adds) has become unnaturally firm and tough, by the interstitial deposit of new fibrous tissue, an impediment to the free passage of blood from it towards the heart, unless it be long continued, will produce but little increase of its size; but it will exert the same, or even greater pressure on the other elements of its texture, and be as apt therefore, or even more apt, to cause secondary biliary congestion."

The size which the liver may attain under these circumstances is remarkable: it sometimes extends downwards to below the umbilicus; anteriorly it forms a swelling evident sometimes to the eye, but always perceptible to the hand, while its enlargement upwards causes it to encroach upon the right lung. It must be borne in mind, however, as observed by Dr. Clendinning,† that the liver may be enlarged, and yet not extend below its normal limits. "A large liver, well sustained superiorly, or strongly pressed upwards by tympanitis, or ascites, might pass (he observes) for small or normal; and a small liver, pushed down by emphysema, or pleuritic effusion above, or by stays outside and around, might pass for abnormally large." The actual size which the congested liver attains may be pretty accurately determined by percussion of the hepatic region. Enlargement of the liver the result of congestion, may usually be distinguished from enlargement of the organ from other causes by the liver preserving its normal shape, which is more or less altered in diseased states of this organ, as well as by the suddenness with which the enlargement supervenes and subsides under treatment. Indeed, the rapidity with which the liver enlarges, and the great increase in size which it attains, has sometimes excited unnecessary alarm in the mind of the practitioner.

When the congested state of the liver has caused considerable

\* Diseases of the Liver.

† Croonian Lectures, Medical Gazette.

increase in size of the organ, the enlargement upwards, which is sometimes from two to three finger-breaths above its normal limits, and as high as the inferior angle of the scapula, or above it, will impede the descent of the diaphragm, and will cause the left side of the thorax to be encroached upon—by which dyspnoea will be aggravated. In addition, the patient usually complains, more or less, of an uneasy sensation in the hepatic region, particularly of a sense of weight: he does not suffer from pain unless pressure is made upon the part, and but seldom even then. When biliary is combined with sanguineous congestion, the secretion of bile may be interrupted, or its escape may be impeded, owing to the pressure exercised upon the biliary plexuses and interlobular ducts, or to the swelling from congestion of the mucous membrane lining the ducts themselves. The patient will suffer from various symptoms of gastric derangement; as thirst, loss of appetite, vomiting, costiveness, or diarrhoea, with colicky pains, followed by jaundice, in a slight or more marked degree, and frequently with diminished secretion of urine. “The gastric derangement which ensues in such cases has a pernicious reaction,” Dr. Furnivall\* observes, “on the original disease, the gastric irritation adding to the cardiac irritation, and aggravating the paroxysms of dyspnoea as well as exciting them; while the retention of those matters which should be excreted from, and by the agency of the liver, deprives us of one of our principal means of relieving the load of labour which is imposed on the heart.” Frequent attacks of, or the long continuance of congestion of the liver, are followed by permanent increase in size of the organ, which becomes harder and more irregular, by congestion of the mucous lining of the alimentary canal, by cachexia, hæmorrhoids, and in aggravated cases, by hæmatemesis, or intestinal hæmorrhage, and when the patient’s constitution has become much enfeebled, ascites generally sets in.

Any form of cardiac disease which directly or indirectly occasions an impediment to the return of the blood ascending by the inferior cava may give rise to congestion of the liver; the forms of cardiac lesion with which it is most frequently associated are:—1st. Considerable contraction of the mitral orifice: here congestion of the lungs always precedes the hepatic congestion. Next in fre-

\* Lancet, 1846.

quency, it occurs in cases where the tricuspid orifice is enlarged, and free regurgitation occurs at each ventricular systole: here the lungs are not engaged, but the venous system generally is engorged. Lastly, congestion of the liver is frequently associated with dilatation of the ventricles, with or without thinning of their parietes; this is frequently combined with a state of the tricuspid orifice permitting free regurgitation; and the congestion is partly due to it, partly to the impediment to the circulation through the heart, and partly to the pressure exercised upon the ascending vena cava, as it is about to enter the right auricle, by the enlarged organ.

Congestion of the liver is occasionally consecutive to lesions of other organs beside the heart; thus it may ensue in any diseased condition of the lungs in which the pulmonary circulation is impeded. Among these, pulmonary emphysema is one of the most frequent. The latter condition itself, however, is no unfrequent accompaniment of diseased states of the heart, and, when congestion of the liver occurs, it may depend as much upon it as upon the emphysematous state of the lungs.

#### CONGESTION OF THE GASTRO-INTESTINAL MUCOUS MEMBRANE.

The portal vein receives the blood from the superior and inferior mesenteric veins, from the splenic and gastric veins; consequently the blood returned from the spleen and pancreas, from the small and large intestines, must pass through the liver before it reaches the right side of the heart. It is easy, therefore, to understand that if congestion of the liver occurs it will react upon the *arrière* current of blood; and, if long continued, that congestion of these organs will ensue. Hence, congestion of the intestines is necessarily a secondary lesion to congestion of the liver, and seldom occurs unless congestion of the liver has preceded it; and may be regarded as purely the result of a mechanical obstacle to the return of the venous blood from these parts.

In the slightest form, where the free return of the venous blood from the gastro-intestinal mucous membrane is merely impeded, the venous trunks simply become congested. When the impediment is greater or the causes have been longer in operation, along with this state of congestion of the veins, "an injection of the small vessels in streaks, stripes, patches, or points, with opacity

of the injected parts, will," Dr. Copland\* observes, "be found; and, in the highest degree, a partial effusion of blood into the sub-mucous cellular tissue, forming ecchymoses, or into the cavity of the part, colouring red the matters contained therein." "The mucous membrane lining the intestines, or even the entire membranes of the intestines, may present," Rokitansky† remarks, "an uniform reddish-black colour, the tissue being saturated with blood, and no injection of blood-vessels being distinguishable: the larger vessels, and particularly the venous trunks, are distended even as far as the mesenterics, and overcharged with blood. In either case hæmorrhage may take place into the cavity of the intestines."

The simple form of congestion, when temporary, is not accompanied by any prominent symptom. When the congestion is more marked, when it has persisted for some time, and when the impediment to the return of the venous blood is greater, the train of symptoms which are laid down as characterizing dyspepsia set in, and the patient may refer all his complaints to the abdominal complication, so that it is possible the diseased condition of the heart might be overlooked. "Congestion generally," as Dr. Williams‡ observes, "impairs the vital properties of internal organs; natural contractility and sensibility are lowered, but pain, spasm, and morbid sympathies are often excited, although in a manner much less distinct and constant than in inflammation or determination of blood."

When the gastro-intestinal mucous membrane has been long or considerably congested, its functions are more or less impaired and its secretions, as the result, may be either increased or diminished, or vitiated; and according as the gastric mucous membrane or that lining the large and small intestines, is particularly engaged, the symptoms will vary somewhat. Thus, when it is limited to the stomach, we may have nausea, vomiting, loss of appetite, and epigastric pain; when the mucous membrane, lining the intestines, is much congested, we shall have flatulent distension, irritation, pain, and constipation, or diarrhoea, according as the secretion is increased or diminished, or altered; when the lower portion of the large intestine is congested, we may have hæmorrhoids in addition.

\* Dict. of Practical Medicine.

† Path. Anat. vol. ii.

‡ Elements of Medicine.

“A congested state of the entire portal system is the least favourable condition imaginable,” Dr. Chevers\* observes, “for active digestion, and proper nutrition; hence, arise flatulent distension of the stomach, (which as it alone will, in some cases, excite symptoms nearly resembling those of angina, invariably produces a most painful interference with a dilated and irritable heart,) and defective assimilation, which, of course, immediately conduces to the deterioration of the fluids, and consequent increase of congestions, dropsical effusions, &c., as well as to the further impairment of every deteriorated organ, including the muscular struction of the weakened and failing heart itself.”

Dr. Dick† is of opinion, that flatulent distention of the stomach may react upon the heart, by its pressure upon the aorta, or ascending vena cava. “Pressure on the latter will,” he observes, “tend to produce a vacuum in the right auricle and ventricle; pressure on the former, will tend to cause distention of the upper part of the aorta, and of the left ventricle.” In this way it is that the apoplexies of full meals are produced; the course of the blood to the lower extremities being impeded by the pressure of a heavy stomach on the aorta, it is forced to the superior part of the body, and the carotids too strongly inject the brain. We must, however, take into account, as Dr. Chevers observes, “the concurrent influence of sudden increase of fluids, from rapid assimilation, in these cases of post-prandial apoplexy.”

The retardation of the circulation in the minute veins and capillaries, and the congestion of the mucous membrane which is the result, may be relieved in part by the transudation of the serous or aqueous parts of the blood, giving rise to diarrhoea with watery stools; or, by the escape of blood itself upon the mucous surface, giving rise to hæmatemesis; frequently by more or less copious discharges of blood from the hæmorrhoidal vessels. The secretion of urine is almost always at the same time diminished, and ascites usually begins to show itself at this period—always, however, preceded by anasarca.

#### CONGESTION OF THE BRAIN.

That a connexion exists between organic disease of the heart and cerebral disease, was noticed so long ago as the time of Morgagni;

\* Treatise on Diseases of the Heart, Calcutta, 1851.

† On Derangements of the Organs of Digestion, 1843.

many clinical observations have been since brought forward to prove it, and abundant evidence of the fact has been, within the last few years, adduced by Dr. Clendinning,\* Dr. Burrows,† Dr. Kirkes,‡ Dr. Law,§ and Dr. Wardrop. |

The circulation in the brain may be disturbed in more than one way, as the result of disease of the heart.—Thus :

1. The blood may be transmitted to the brain in increased amount, or with undue force, by an hypertrophied left ventricle.

2. There may be an impediment to the free return of the venous blood from the brain to the right side of the heart, owing to disease in the latter organ.

3. The blood may not undergo the necessary changes in the lungs, the effect of disease of the valves or orifices of the heart, when a mixture of venous and arterial blood will circulate through the brain.

4. The blood may be transmitted to the brain in deficient quantity, owing to obstructive disease of the orifices at the left side of the heart, as pointed out by Dr. Law.

5. A part of the brain may be insufficiently supplied with blood, the effect of obstruction of one of the cerebral arteries by a portion of fibrin detached from a diseased valve, as pointed out by Dr. Kirkes.

Lastly.—Independent of disease of the heart, the blood itself may be thin, watery, and deficient in some of its constituents, as in anæmia ; or it may be rich and quickly formed, as in plethora.

Writers upon diseases of the heart, have laid considerable stress upon the influence of hypertrophy of the left ventricle, in causing cerebral congestion, by its transmitting the blood in increased quantity or with augmented force, to the brain. The arteries which supply the brain run a short course from the arch of the aorta, and they receive, almost immediately, the force and the impulse of the left ventricle ; the circulation in the head and face is consequently active, indicated by the greater heat of the parts than of the extremities. Hence, we should expect, that when the left ventricle is hypertrophied and dilated, cerebral congestion would be likely to ensue ; but in most instances, hype

\* Croonian Lectures, Med. Gazette.

§ Dub. Jour. of Med., vol. xvii.

† Disorders of the Cerebral Circulation.

|| Treatise on Diseases of the Heart

‡ Med. Chir. Trans. vol. xxxv.

trophy of the left ventricle is itself a secondary affection, the result of some impediment to the passage of the blood through, or out of the chambers of the heart. Hence, although the heart's action may be strong, the pulse is often weak, showing that the strength of the current is not necessarily augmented, and that the amount of blood transmitted by the ventricle is not always increased under such circumstances.

In considering congestion of the liver, we found that one of its effects was to cause an increase in the size of the congested part; and, we can readily understand that such a delicate organ as the brain, which is enclosed within solid bony walls, could not undergo even slight enlargement, without the cerebral substance and nerves suffering compression. That injurious effects do not more frequently ensue, has been explained by Dr. Burrows, as follows:—1st “Owing to the great development of the venous system within the cranium and spinal canal, which affords a ready exit for the redundant blood;” and 2dly “Owing to the existence of the cerebro-spinal fluid.” This fluid he has shown to play a most important part, as—“by its capability of oscillation between the ventricles of the brain and the spinal canal, it compensates for variations in the quantity, or tension of the circulating fluids of the brain. When by increased action of the heart, more blood than common is thrown upon the cerebral vessels, the cerebro-spinal fluid subsides into the spinal canal, and injurious pressure upon the cerebral pulp is thus, within certain limits, abated; and on the other hand, if less blood than usual pass into the brain, the fluid rises into the cranium, and the equilibrium of tension is preserved.”

Although cerebral congestion might be the result of hypertrophy of the left ventricle, this state is even more likely to ensue if there is an impediment to the free return of the venous blood from the brain. In a state of health, this fluid readily returns from the head, being assisted by gravity, and anything which interferes with it is quickly felt; thus, if the head remains for any length of time on a plane lower than the body, symptoms of cerebral plethora will ensue; as the internal jugular veins are not provided with valves, there is no provision to prevent the reflux of their contents towards the brain. Indeed, persons partly asphyxiated by submersion, or in a state of stupor from intoxication, have often



been deprived of life by being carelessly removed in a position which allowed the head to hang downwards.

When the circulation in the brain is disturbed, from any of the causes mentioned, the patient may suffer from headache, vertigo, tinnitus aurium, or ocular spectra. "Many who are suffering from a permanent disorder of the heart, have," Dr. Wardrop observes, "peculiarities in their sight, which are caused by changes in the circulation within the head, so that certain disturbances in the functions of the retina become as important diagnostic *symptoms* in disease of the heart as those which take place in the organ of hearing." These are "false images, or the appearance of bodies floating before the eyes, sparks of fire, dimness of sight even to blindness; all which symptoms may either exist separately and in one or both eyes, or they may be combined with other disturbances in the cerebro-spinal system." In some instances, disagreeable sensations in the head of rushing, beating, or throbbing are experienced by the patient; in others, the sensation is rather of weight, tightness, or heaviness. In some instances, insomnia is complained of, or if the patient sleeps his rest is disturbed by frightful dreams; in others, drowsiness or somnolency are more marked. The face may be flushed, the lips and cheeks congested or, on the other hand, it may be very pale. When the disturbance of the cerebral circulation is more considerable, the drowsiness or somnolency may be succeeded by coma; or the insomnia by epileptic or hysterical convulsions. In some instances as Dr. Furnivall observes, "headache is early complained of, and is both severe and long continued. This is doubtless caused by cerebral venous congestion, and it and other cerebral symptoms have been sometimes so prominent as to mask and to draw attention from the original and originating disorder." When the venous blood is not entirely converted into arterial in the lungs, a mixture of venous and arterial blood is conveyed to the brain evidenced by drowsiness and stupor, or by wandering of the mind and a transitory and sometimes pleasing kind of delirium. The latter results are not, however, observed as often as might be expected; the majority of those who die of cardiac disease preserve their intellects perfectly to the close.

Dr. Wardrop in his recent valuable treatise,\* dwells at consi-

\* On Diseases of the Heart, 1851.



derable length upon this part of the subject. He is of opinion that many anomalous symptoms, and many affections, to which the name *nervous* is usually applied, depend upon a primary disorder of the heart; and he has given the following *table* of the symptoms which may arise from irregularities, or inequalities, or other alterations in the supply of blood to the cerebro-spinal system, the result of functional, or organic disease of the heart:

Watchfulness—*insomnis*.

Somnolency—*sepor*.

Coma.

Fainting—*syncope*.

Dreaming, sleep-walking, nightmare.

Headache.

Mental Irritability.

Mental Aberration.

Giddiness—*vertigo*.

Ringing in the ear—*tinnitus aurium*.

Ocular Spectra—*musci volitantes*.

Neuralgia.

Angina.

Palsy—*hemiplegia-paraplegia*.

Apoplexy.

Epilepsy.

Catalepsy.

Hysteria.

Convulsions—*chorea—subsultus—tremors*.

Apoplexy and paralysis are regarded, by several writers, as direct results of hypertrophy of the left ventricle. Dr. Burrows says, "hypertrophy of the left ventricle must be admitted as a powerful predisposing, or even exciting cause of apoplexy and sudden hemiplegia." Dr. Hope, in the "Cyclopædia of Medicine," and subsequently in his "Treatise on Diseases of the Heart," observes: "Instances of apoplexy supervening upon hypertrophy have been so frequently noticed, that the relation of the two, as cause and effect, is one of the best established doctrines of modern pathology. Eight or nine cases of suddenly fatal apoplexy, and numerous cases of palsy from hypertrophy, have within a few years fallen under our observation; whence we are led to the conclusion, with MM. Richerand and Bertin, that hypertrophy forms a stronger predisposition to apoplexy than the apoplectic condition itself."

It appears to me, however, that the influence of hypertrophy of the left ventricle, in causing sanguineous apoplexy or hemiplegia, has been overrated; for though in many instances where these affections terminate fatally in individuals advanced in life, the heart is found to be enlarged, we generally find the coats of the arteries of the brain to be likewise diseased; besides, we know, that, in the majority of cases, hypertrophy of the left ventricle

is itself a secondary affection, the result of some impediment to the circulation—"Arterial cerebral apoplexy not unfrequently," Dr. Chevers\* observes, "occurs in *association* with hypertrophy of the heart. The same causes, especially an excess of circulating fluid, tend to produce both; and the state of the heart may, in some cases, be immediately operative in occasioning the effusion." "I believe, however (he adds), that this accident occurs far less frequently than is generally supposed." Corvisart goes even still further, he says he never saw organic disease of the heart directly occasion apoplexy and death. "I have," he says, "many times in cases of this kind, seen all the vessels of the brain, and particularly the sinuses, gorged with blood, but I have never met with extravasation of blood either in the substance of the brain, or in its cavities." "In several of the cases (he adds) related elsewhere, the death was sudden; I cannot, however, assert that I have ever seen a single case of apoplexy which was evidently the result of cardiac disease."

It is not usually, as M. Lallemand† remarks, in the apoplectic cases of heart disease, that we observe the livid lips and cheeks, or the œdema, which point to obstruction of the circulation in the veins." "It is in comparatively early cases," as Dr. Ormerod‡ observes, "where the general symptoms of heart disease are scarcely developed, that sanguineous apoplexy most commonly occurs; not in those patients whom a cold winter sends into our hospitals, loaded with dropsical accumulations and with venous blood—cold, livid, and struggling for breath. These are rather the subjects of softening of the brain or of serous effusion, than of sanguineous apoplexy."

*Deficient supply of blood to the Brain.*—Dr. Law§ was the first to call attention to the injurious effects of a deficient supply of blood to the brain, as a result of cardiac disease. "Pathologists hitherto," he observes, "have almost entirely confined their attention to the excess of the normal quantity of blood, or the congestion produced in organs by disease of the heart, but have overlooked the deficiency that must necessarily result to other organs, and consequent hurt to their nutrition and function. Thus

\* Treatise on Diseases of the Heart, Calcutta, 1851.

† Recherches sur l'Encéphale.

‡ Gulstonian Lectures, Med. Gaz.

§ Dub. Jour. of Medicine, May, 1840.

in disease so affecting the mitral valve as to lead to a considerable narrowing of the opening between the auricle and ventricle, exclusive attention has been directed to congestion of the lungs, liver, &c., and no notice has been taken of any ill effects resulting to other organs, from their supply of blood being proportionably diminished." In the valuable paper from which the foregoing is an extract, Dr. Law has recorded cases, showing that ramollissement of the brain may occur in connexion with diseases of the heart, whose effect is either directly or indirectly to diminish the supply of blood to the brain; and that this cerebral lesion may be connected with disease of either the mitral or aortic valves or orifices. It is most common in cases of considerable contraction of the mitral orifice; next, in obstructive disease of the aortic orifice; rarely in cases of mitral regurgitation. Dr. Fleming\* has, however, recorded one where semi-apoplectic attacks, with temporary hemiplegia, occurred in connexion with a very dilated state of the mitral orifice, permitting free regurgitation through it.

When the parietes of the left ventricle are softened or attenuated, or have undergone fatty degeneration, the weakened ventricle is incapable of propelling its contents with sufficient force through the system; and the brain may, under such circumstances, be insufficiently supplied with arterial blood. Those syncopal attacks and semi-apoplectic seizures which sometimes occur in this condition of the ventricle, and which leave no trace of paralysis behind, are probably due to this cause.

Recently, Dr. Kirkes† has brought forward evidence that softening of a portion of the brain (from imperfect nutrition) may result from obstruction of a main cerebral artery by the arrest of a portion of fibrin in its canal, detached from a vegetation upon the valves at the left side of the heart, and conveyed by the circulating blood. The artery which was obstructed in his cases, was the middle cerebral; which he considers the most likely to arrest a portion of fibrin floating in the blood transmitted to the brain by the internal carotid artery. "Once arrested at the angle or within the canal of the middle cerebral artery, a mass of fibrin, if large enough to block up the vessel, becomes," he observes, "at

\* *Dub. Jour. of Medicine*, vol. xvii.

† *Med. Chir. Trans.* vol. xxxv.

once the cause of loss of function, and subsequent atrophy, to almost all that portion of the brain supplied by the obstructed vessel; for, although by the arrangement of the vessels composing the 'circle of Willis,' ample provision is made against obstruction ensuing in any of the main arterial channels of either side, previous to their arrival at the circle, there is comparatively little provision for an obstruction ensuing in any of the main branches into which this arterial circle breaks up. This remark applies especially to the middle cerebral artery, which if plugged up at its origin, becomes at once almost useless as a blood-vessel; for nearly all its divisions, especially those for the central parts of the brain, proceed to their several destinations without receiving any anastomosing branch from the other divisions of the 'circle of Willis.'"

Dr. Kirkes believes that his cases establish the following conclusions: 1. "That softening of a portion of the brain, with attendant loss of function, might result from obstruction of a main cerebral artery by the lodgment of a plug of fibrin within its canal." 2. "That the foreign substance thus obstructing the vessel is probably not formed there, but is derived directly from warty growths situated on the left valves of the heart." He thinks it, likewise, not improbable, that "many cases of partial and temporary paralysis suddenly ensuing in one or more limbs of young persons, especially if accompanied by signs of cardiac disease, might be due to interruption of a proper supply of nutriment to the brain by the temporary plugging up of a principal cerebral artery by fibrin, detached from a diseased valve on the left side of the heart."

#### POLYPOID CONCRETIONS IN THE CAVITIES OF THE HEART.

The term *polypi* applied to the concretions which occasionally form in the cavities of the heart is rather an unfortunate one, these bodies having no analogy whatever with polypi, either in composition, or in the manner in which they are developed; while the views which have been advanced respecting them appear to be in several particulars erroneous.

Dr. Hope describes three species, viz.: 1. Unorganized polypi 2. Slightly organized. 3. More completely organized. M. Bouillaud likewise makes three species; his description pretty near

coincides with that given by Dr. Hope. These concretions, it appears to me, may be all included under the four following heads: viz. 1. Concretions consisting of a coagulum coated with fibrin. 2. Concretions composed exclusively of fibrin. 3. Concretions composed exclusively of lymph. 4. Concretions consisting of lymph coated with fibrin.

1. *Concretions consisting of a coagulum coated with fibrin.*—These (which, however, scarcely deserve the name inasmuch as they are either a post-mortem occurrence, or are formed only during the last hours of life,) consist of a more or less firm coagulum of blood, partially or completely coated with fibrin, which fills and distends one or both the cavities of the right side of the heart particularly, frequently extending into the large vessels, but not adherent to the parietes. They occur in cases in which the blood had accumulated during the last hours of life in the right cavities of the heart; and their occurrence is to be regarded rather as a pathological phenomenon than as a circumstance of any practical importance.

The three other forms are developed during the life of the patient, are accompanied usually by sufficiently well-marked symptoms, and are frequently the immediate cause of death; whether the diseased state under which they arise have its seat in the heart itself, in the lungs, or in some other part.

2. *Concretions composed exclusively of fibrin.*—These which are the most frequent, have a yellowish-white colour, are semi-transparent, more or less elastic, and more or less adherent to the parietes, particularly to the carneæ columnæ and tendinous cords of the valves. They occur in cases, where a considerable impediment to the circulation had existed for some time previous to the patient's death; whether this impediment was seated in the heart or in the lungs; in consequence of which, the fibrin separates from the other constituents of the blood, and concretes upon the valves or orifices, or upon the parietes of the ventricles or auricles. These fibrinous concretions may be developed in any disease in which the circulation through the cavities of the heart is much impeded. They are met with in acute bronchitis, in pneumonia, and in the advanced stages of valvular disease; and they are not unfrequently the immediate cause of death in these diseases.

3. *Concretions composed exclusively of lymph.*—These, which

are the most rare, are altogether different in their origin from the preceding; while the former are developed by the mechanical separation of the fibrin from the other constituents of the blood in its transit through the heart, the latter are deposited by the vessels of the part. This form of concretion occurs in cases of inflammation of the lining membrane of the heart; it usually has its seat upon the valves or orifices of the left side of the heart and it is deposited by the vessels of the part the seat of the inflammation: in fact, as it is derived from the vessels which supply the tissue of the heart with blood, its source is as different as is the blood which circulates through the heart, from that which supplies nutriment to the organ.

4. *Concretions consisting of lymph coated with fibrin.*—This form of concretion is a combination of the two last; it is more frequent than that last described, but more rare than the two former species. In it, the nucleus is lymph, which is deposited upon the valves or orifices as the result of endocarditis; but the great bulk of the concretion consists of fibrin deposited upon it, sometimes in concentric layers, as we see in aneurismal sacs. This form of concretion occurs in cases where the lining membrane of the heart had been at a former period the seat of inflammation and where an impediment to the circulation through this organ had existed for a longer or shorter time previous to the patient's death. Like the last species, it is found in the left cavities of the heart.

These four forms of concretion are not all of equal frequency; some are sufficiently common, others are exceedingly rare; some are usually only found upon the right side of the heart, others only upon the left. The concretions composed of coagulated blood or of fibrin are most frequent in the right cavities of the heart, and often extend through the auriculo-ventricular orifice or into the large vessels. Those which consist of lymph, or lymph coated with fibrin, are most frequent in the left cavities of the heart. The former are sometimes of such a size as to obstruct and distend the cavities of the heart; the latter, in general, merely impede the action of the valves. When composed of coagulated blood or of fibrin, they are smooth upon the surface, and mould to the shape of the cavity in which they are contained; while those composed of lymph exclusively, their surface is uneven and the

shape irregular. The concretions consisting of coagulated blood are not adherent, though they may be entangled among the *carneæ columnæ*, or *cordæ tendinæ*; those which consist of fibrin are more adherent, but may still always be detached; those composed of lymph, or in which the nucleus consists of lymph, are adherent to the part upon which they are deposited.

The concretions which consist of fibrin are sufficiently frequent, and the opinion has been advanced on very respectable authority, that these fibrinous masses not only become organized, but that they undergo the same changes as organized tissues in other parts, such as softening, the formation of pus, &c.; and that they are sometimes united by cellular tissue to the lining membrane of the cavities of the heart. Thus, Bouillaud, describing them, observes: "They are white, decolourized, elastic, glutinous, adherent to the parietes of the heart, and confused with the tendinous cords and fleshy columns. They are in some sort half-organized, and very analogous either to the inflammatory crust of the blood, or to false membranes. Some present here and there points, or red lines, which are really nothing more than the rudiments of vessels." Dr. Hope observes, speaking of the slightly organized polypi: "It may be premised as a fact ascertained by observation, that *fibrin* separated from the blood, and become concrete in a living organ (whether *the heart, the blood-vessels*, or serous, cellular, or other tissues into which it had been extravasated), retains its vitality, and is susceptible of organization in an equal degree with *inflammatory lymph*." This view of the subject is evidently incorrect: these masses of fibrin are unorganized, and incapable of becoming organized; nor are they ever united to the lining membrane of the heart by cellular tissue. Indeed, a knowledge of their composition, and of the manner in which they are formed, ought to have been sufficient to set aside such an absurd idea, consisting as they do simply of one of the constituents of the blood, mechanically separated from the others in its transit through the heart.

The concretions which consist of lymph, instead of being derived from the blood which circulates through the heart, are deposited by the vessels of the inflamed tissue, and being analogous to the false membranes secreted by serous surfaces, may evidently become organized: in fact, the vegetations found upon the valves



and orifices of the heart, consecutive to endocarditis, are nothing more than these concretions upon a small scale. Deposits of lymph in the cavities of the heart, of such a size as to deserve the name of polypoid concretions, are however very rare; more frequently the nucleus is lymph, and the great bulk of the concretion is made up of fibrin, deposited upon it either in amorphous masses or in regular stratified layers. In some instances, however, such an amount of lymph is secreted as not only to impede the action of the valves, but to obstruct the circulation through the cavities of the heart, and to occasion the death of the patient, an example of which was communicated by me, some time since to the Surgical Society of Ireland; and the preparation is now in the Museum of the College of Surgeons.

The symptoms of polypoid concretions in the cavities of the heart, are such as might be expected to arise from an impediment to the passage of the blood through the central organ of the circulation, and the intensity of the symptoms will of course be proportionate to the size of the foreign body and the rapidity with which it is formed. As the coagula coated with fibrin, which have been classed by writers with these formations, appear not to be formed during life, we may omit their consideration here. When these concretions are developed rapidly, and when they quickly attain a considerable size, they give rise to a most intensely severe and distressing train of symptoms, most agonizing to the sufferer, and most distressing to witness. These have no reference to the lungs rather than to the heart: they are dyspnoea suddenly arising accompanied by a feeling of impending suffocation and a distressing sense of oppression; with which are associated sleeplessness, anxiety, restlessness, inability to assume the recumbent posture, coldness of the surface, congestion, or extreme paleness of the face. The pulse is small, feeble, and hurried, or it is imperceptible, although the action of the heart may be strong. In two cases,\* related elsewhere, in one of which the concretion occupied both ventricles, there was no intermission or irregularity of the pulse, nor any abnormal murmur accompanying the heart sounds; neither was there any increased extent of dulness in the præcordial region, nor was the action of the heart confused.

\* Dublin Medical Press, vol. xxi.



irregular: symptoms which have been laid down by writers as almost pathognomonic.

The diagnosis must always be one of some difficulty, particularly if we have not seen the patient previous to the occurrence of the symptoms due to their formation; the difficulty will be somewhat less if the patient had been for some time under observation, when, if the train of symptoms enumerated suddenly set in, the formation of a polypoid concretion in the heart is probable, provided the disease under which the patient labours is such as to impede the circulation through the lungs or heart; and the diagnosis will be confirmed if there is no evidence either of large effusion of fluid into the pericardium or pleura; or, of the secretion of mucus into the bronchial tubes, either in such a quantity as to impede the entrance of air, or of so viscid a nature as to obstruct the passage of air into the lungs.

The development of these concretions in the cavities of the heart, particularly when they are of such a size as materially to interfere with the circulation through this organ, is usually quickly followed by the death of the patient. With respect to treatment, it is clear that we can do little more than palliate symptoms, and support the powers of life. As to bringing about their resolution, the idea seems too absurd almost to be entertained; nevertheless, we find writers recommending the administration of substances supposed to be capable of dissolving polypi, or of rendering the blood more fluid, such as the salts of potass, or diluent drinks given freely; while others, among whom M. Bouillaud is the most conspicuous, consider that bleeding is the best measure that can be employed when these concretions have once formed.

The following conclusions appear to be deducible from the preceding details:

1. That the concretions which occasionally form in the cavities of the heart during life, and impede the action of the valves, or obstruct the passage of blood through this organ, although termed polypi, have no analogy whatever with polypi, either in appearance, composition, or mode of development.

2. That these concretions may consist either of fibrin, or of lymph, or of lymph coated with fibrin. Those composed of fibrin are most frequent upon the right side of the heart, but may occur

on both sides; those which consist of lymph, or of lymph coated with fibrin, are usually found only on the left side of the organ.

3. That the concretions composed of fibrin, whether they occur in amorphous masses, or in stratified layers, are mechanically separated from the blood which circulates through the heart. That the concretions composed of lymph, whether this forms the substance of the mass or merely its nucleus, are deposited by the vessels which supply the heart itself with blood.

4. That fibrin, whether it constitutes the substance of the concretions, or whether it is deposited in concentric layers in the sac of an aneurism, is perfectly unorganized, and perfectly incapable of becoming organized; that lymph, on the contrary, is an organizable substance.

5. That where pus has been found in fibrinous concretions contained in the heart, its presence is to be regarded as the result of phlebitis, not as a product of inflammation in a substance which being unorganized, is incapable of undergoing such change.

6. That increased extent of dulness in the præcordial region, confused or irregular action of the heart, intermission or irregularity of the pulse, or an abnormal murmur accompanying the heart's sounds, are not necessarily symptoms of the development of these concretions.

7. That no means are known by which polypoid concretions in the heart, once formed, can be dissolved; consequently, the administration of substances which render blood, when removed from the body, more fluid, or which are supposed to be capable of dissolving them, can have no effect.

#### CARDIAC DROPSY.

Dropsy is a common secondary symptom of the advanced stage of cardiac disease, and usually also a late symptom. It makes its appearance as œdema or anasarca, and this may be the only form up to the close. The dropsical effusion, which is partial and slight at first, commences in some instances with a puffiness of the eyelids, more frequently as œdema about the ankles; in slow degrees it ascends towards the trunk, and ultimately often engages the upper extremities and face. When general, the parts in which the cellular tissue is lax, as the scrotum in men

and the labiæ in females become sometimes enormously swollen ; and as the patient at this period is usually confined to bed (owing perhaps to the unwieldy state of the lower extremities), the infiltration predominates in the most depending parts, the posterior surface of the trunk when he lies upon the back, or one side of the face, or one hand, or arm when he lies upon his side. Towards the close, effusion very often takes place into one or both pleura, increasing the dyspnoea, aggravating materially the patient's sufferings, and hastening the fatal result. Ascites to any very great extent, on the other hand, is uncommon.

The first appearance of dropsy in cases of organic disease of the heart marks, as Dr. Latham\* observes, "an eventful period : it marks the period when a new law is beginning to take effect in the circulation, and to gain a mastery over the law of health. The law of health (of which the sound heart is the prime agent) retains the blood within the blood-vessels, or dispenses it only for the needs and uses of health. The new law (of which the unsound heart is the prime agent) suffers, or forces the blood, or some of its constituents to escape, and to form accumulations of serum out of the courses of health. A little œdema of the ankles, or a little œdema of the face, is a sufficient notice of the first yielding of the blood-vessels to this new law, which is no other than a mechanical necessity, against which they can no longer hold out. It is the earliest beginning of serous effusion, which may go on increasing until it has pervaded the entire cellular structure, and filled every serous cavity of the body."

The theory of the production of dropsy in cardiac disease is sufficiently simple : the return of the venous blood to the right side of the heart being impeded, general venous congestion ensues ; after a time, the over-distended state of the capillaries and minute veins is relieved by the exudation or transudation of the thinnest part of the blood (the serum or liquor sanguinis) into the subcutaneous areolar tissue. This, as a matter of course, occurs first in the most dependent parts, and the lower extremities consequently are its earliest seat, because the returning blood has to ascend in them against gravity. No fact is better established than that local anasarca will follow obstruction to the return of the blood by the veins of a limb ; thus the œdema of the lower ex-

\* Lectures on Diseases of the Heart, vol. ii.

tremities in advanced pregnancy has its cause in the pressure the gravid uterus, upon the large veins which return the blood from these parts. Again, aneurismal or other introthoracic tumours, which compress the superior cava, occasion first distension of the large veins above, and eventually anasarca of the face, neck and upper extremities. The fact has also been demonstrated by direct experiments upon animals; thus Lower placed a ligature upon the inferior cava of a dog, which was followed by anasarca of the posterior extremities; in another, he tied the jugular vein and anasarca ensued in the parts above the ligature.

Although the immediate cause of cardiac dropsy, as a general rule, is some mechanical impediment to the return of the venous blood to the right side of the heart, there are certain circumstances which favour its occurrence. Thus, in organic disease of the heart, as well as in other chronic diseases, the blood undergoes changes which predispose to dropsy. These, as MM. Beoque and Rodier\* have shown, consist in the diminution of the globules, fibrin, and albumen, and increase in the watery portion of the blood. The diminution of the fibrin favours the occurrence of hæmorrhage from mucous surfaces; the diminution of the albumen of the serum of the blood leads more particularly to dropsy. Again, we know that in cases of general venous congestion, the liver or the kidneys are liable to become engaged by which the predisposition to the occurrence of dropsy is materially favoured. In many instances, likewise, disease of the right side of the heart is the result of long-standing disease of the lungs, particularly chronic bronchitis with emphysema, which itself is capable of giving rise to dropsy of the cellular tissue, and of the serous cavities.

Cardiac dropsy, properly speaking, occurs only in cases of organic disease of the heart; it is almost always preceded by general venous congestion, and it is to be regarded as an effort of nature to relieve the over-distended state of the vessels, when a considerable and permanent impediment exists to the return of the venous blood to the right side of the heart. Cardiac dropsy is most common in valvular disease; and the forms of valvular disease to which it is met with, in the order of their frequency, are :

\* L'Union Médicale.

1. Considerable contraction of the left auriculo-ventricular orifice.
2. Dilatation of the right auriculo-ventricular orifice, with hypertrophy and dilatation of the right ventricle.
3. A state of the mitral valve and orifice permitting free regurgitation.
4. Considerable contraction of the aortic orifice.

Thus, when the mitral orifice is much contracted, there is a permanent impediment to the passage of the blood from the left auricle into the left ventricle, the auricle becomes distended, and the pulmonary veins are unable to empty themselves; congestion of the pulmonary tissue and of the bronchial mucous membrane follows, which may be relieved for a time by artificial means, or spontaneously by increased secretion from the bronchial mucous membrane. At length, the right cavities of the heart suffer, the auricle becomes dilated, and the ventricle hypertrophied, the right auriculo-ventricular orifice participates in the dilatation, and the tricuspid valve permits free regurgitation; the *venæ cavæ* are thus prevented from unloading themselves, general venous congestion ensues, followed sooner or later by dropsy.

When the left auriculo-ventricular orifice is dilated, or when the mitral valve is diseased, so as to permit free regurgitation into the left auricle at each ventricular systole, the same results follow, but in a less marked degree than in cases of extreme contraction of the orifice. Dr. Blackiston,\* however, looks upon it as the most frequent cause of pulmonary congestion.

Dilatation of the tricuspid orifice, with hypertrophy, and dilatation of the right ventricle is one of the commonest secondary effects of obstructive or regurgitant disease at the mitral orifice; but it also frequently occurs independent of either, and it is an ordinary effect of chronic bronchitis with emphysema. When this orifice is dilated, whatever be its cause, and when much of the contents of the right ventricle regurgitates into the auricle at each systole, particularly if the ventricle is at the same time hypertrophied, the return of the blood by the *venæ cavæ* to the right auricle will be materially impeded, which will react upon the venous system throughout the body, general venous congestion will ensue,

\* On Diseases of the Chest.

followed, sooner or later, by dropsy, just as in the cases where the impediment to the circulation commenced at the left side of the heart.

Dropsy, in my experience, does not supervene so early, and when it does occur, the anasarca is neither so extensive nor prominent a symptom in regurgitant disease of the mitral orifice as in a contracted state of this orifice; it is also more amenable to treatment in the former than the latter. Dropsy to any extent is rare in regurgitant disease of the aortic valves, indeed many cases of this form of valvular lesion terminate fatally either without the occurrence of dropsy, or with merely œdematous swelling of the ankles.

The period which intervenes, in cardiac disease, before the occurrence of dropsy is very variable, and depends upon a variety of circumstances; such as the part of the heart engaged, the nature or amount of the cardiac lesion, and its cause; the age of the patient, and the state of his health; whether he is debilitated or anæmic, or the subject of pulmonary, renal, or hepatic disease; the habits of the patient, whether he is temperate or intemperate; his occupation or trade, whether it is healthy or otherwise, or whether it is one in which much muscular exertion is required or the contrary; his station in life, whether it is such as to place him above the necessity of labouring for his daily bread, exposed, perhaps, to all the vicissitudes of the weather, or whether it is such as to save him from experiencing the ill effects of insufficient nutriment, or the contrary. As a general rule, almost, dropsy supervenes earlier, the earlier that general venous congestion ensues; as a general rule, likewise, it sets in sooner among the labouring orders than the better classes, although the kind and amount of cardiac lesion may be the same in both. Indeed, as Dr. Ormerod observes, "The history of patients (suffering from dropsical effusion in connexion with cardiac disease) who seek admission in our hospitals, commonly speaks of exposure to cold, or distress, or of unusual exertion."

Cardiac dropsy is at first amenable to treatment in the great majority of cases. We have constantly patients in hospital labouring under heart disease, and dropsy; the dropsical effusion is removed by treatment, and the patient returns to his occupation.

\* Gulstonian Lectures, Medical Gazette, 1851.

before long, however, it recurs, he again seeks admission into hospital; and this not unfrequently happens several times. Eventually, remedies fail to produce the same effects; renal is superadded to cardiac dropsy, the patient's strength fails, his constitution gives way, and he sinks under the disease—death being very generally preceded by effusion into the pleura upon one or both sides, or being hastened by intercurrent attacks of pneumonia. In some rare instances, the anasarca has disappeared entirely a short time before death.

The urine in cardiac dropsy is sometimes highly albuminous, although no disease of the kidneys exists. The mode in which albumen finds its way into the urine in these cases, has been pointed out by Dr. O'Beirne.\* We have seen that venous congestion always precedes anasarca; now, when the ascending cava is prevented from returning its contents freely into the right auricle, and the circulation in it is impeded, the renal veins, being unable to empty themselves, become distended, and ultimately the renal circulation is relieved by the exudation of the serous portion of the blood into the infundibula of the kidneys, which mixing with the urine causes it to afford evidence of the presence of albumen when tested, and according to the amount of the serum, the urine will either yield merely a trace of albumen, or will be highly charged with it.

The presence of albumen in the urine is sometimes, however, really indicative of renal disease; thus, when the impediment to the circulation has been of long duration, if the patient has suffered several attacks of dropsy, and congestion of the kidneys has lasted for some time, renal comes to be superadded to cardiac dropsy, and the urine on examination presents evidence of the presence of albumen. Dr. Miller† says "he has rarely seen general dropsy occupying the entire superficial areolar tissue, which was ascribed to cardiac disease, that had not conjointly a renal origin."

The anasarca which accompanies the advanced stage of some forms of valvular disease, particularly considerable contraction of the mitral orifice, is usually more extensive, the swelling and distension are greater, and the pain is more severe than when anasarca supervenes upon other diseases. The lower extremities occasion-

\* Dublin Medical Press, vol. viii.

† The Pathology of the kidney in Scarlatina.

ally attain an enormous size, and the sufferings previous to the fatal termination are sometimes extreme, and protracted. The integuments, too, are liable to run into gangrene, and this is still more likely to occur, if we are induced to give exit to the serum by punctures in the legs, or upon the dorsum of the foot, although a fine needle be employed for the purpose; inflammation of an erysipelatous character running into gangrene quickly ensues, accompanied by intense pain, and soon terminating life.



## CHAPTER IX.

**CARDIAC DISEASE.—PROGRESS AND DURATION OF CARDIAC DISEASE.—TERMINATION OF CARDIAC DISEASE.**

cardiac, as of other diseases, may be either indirect, disposing ; or direct, immediate, or exciting ; many however, in the present instance, partake likewise of the latter. Among the indirect or predisposing are enumerated hereditary predisposition, the rheumatism, pre-existing morbid states of other organs, of the body, particularly of the blood ; the sanguine and plethoric habit, a particular conformation of body, occupations or trades, which either require long continued unusual muscular exertions, or which impede the motions of the diaphragm, and interfere with the motions of the lungs, and excesses of all kinds, and powerful emotions, whether of an exciting or depressing nature.

Immediate, or exciting causes of cardiac disease have been classified by systematic writers ; by some they have been divided under three heads, viz. the “extrinsic, the intrinsic, and the accidental.” Dr. Copland (whose classification is the most recent), makes four sub-divisions, viz. the “mechanical, the physical, the moral, and the pathological.” By these have been arranged according as the disease has its origin, in abnormal states of the blood, the lungs, the vessels, or the heart itself.

However, as the same disease of the heart may arise under different circumstances, and as any one morbid condition may be the result of several distinct causes, and when once established may itself be the cause of several other morbid states, it is difficult to lay down any classification of the kind which is the most useful. For instance, a diseased state of the aortic valves, or any impediment to the current of the

blood at a distance from these valves, may be the immediate cause of hypertrophy of the left ventricle; prolonged physical exertion or the habitual indulgence in intoxicating liquors to excess, may be followed by the same result; any of them, however, in place of causing hypertrophy, may give rise to dilatation of the same ventricle; and any of them might be in operation for a considerable time without being followed by either; moreover, disease of the aortic orifice or of its valves, is itself the result of previous disease and may be due to several different causes.

Corvisart, in the chapter of his work in which he treats of the causes of heart disease, quotes the well-known truism—"the first day of life is but the first step towards the grave." The machine which carries on the functions of the animal economy in which life consists, as we know not destined to last for ever, it contains within itself the elements of decay, and this applies with peculiar force to the heart, no other organ performing such unceasing duties;—functions which cannot be disturbed without every other organ sympathizing, and motions which cannot be interrupted even for a few seconds without death ensuing. In addition, the heart above all the other organs is most readily acted on by mental impressions, emotions of the mind the most opposite, the exciting as well as the depressing passions, hurrying, increasing or otherwise disturbing its action. Hence, we ought rather to be surprised (as Corvisart remarks) that the heart is not more frequently diseased than it is.

It sometimes happens that the heart is malformed at birth; there is a deficiency in some part, or an irregularity in the origin of the large vessels which spring from it (the aorta and pulmonary artery), in consequence of which a mixture of venous and arterial blood is frequently permitted. Omitting, however, for the present the consideration of these congenital malformations, which constitute a distinct and original source of disease, there can be no doubt that some individuals are born with a predisposition to disease of this organ, its parietes being thinner than natural, thicker; or the whole organ does not bear that proportion to the rest of the body which it should in a well formed subject, being either too large or too small for the capacity of the chest.

Again, the relative proportion between the heart and the capacity of the chest may be altered, owing to contraction

other deformity of the parietes of the latter, the result of disease setting in early in life. In such subjects, frequent or protracted physical exertion, or the continued and habitual use of stimulants will be much more likely to be followed by disease of the muscular parietes, or of the valves, or by dilatation of the cavities of the heart, than if the same causes came into operation in an individual in whom no such predisposition existed.

In a great many instances, cardiac disease may be traced, either immediately or remotely, to inflammation of the investing, but particularly of the lining membrane of the heart, whether this has its cause in acute rheumatism, which is by far the most frequent, or in exposure to cold ; or whether it accompanies morbid states of the blood, or of the general system ; yet, this most fruitful source of cardiac disease was scarcely recognised as a cause until within, comparatively, a few years. Injuries of the left side of the chest, such as blows, contusions, or falls, probably by giving rise to inflammation, are likewise occasional causes of cardiac disease ; I have seen several cases where it could be distinctly traced to a blow or fall, or to a severe contusion of the part : these were all young subjects.

Morbid conditions of other organs occasionally react upon the heart, and may thus become an exciting cause of disease. For instance, chronic bronchitis with emphysema of the lungs, by obstructing the pulmonary circulation, gives rise, first, to distension of the right chambers of the heart, followed by dilatation of the right ventricle, usually with, sometimes without hypertrophy of this ventricle. Indeed, Dr. Taylor\* is of opinion, that pulmonary obstruction may cause not only enlargement of the right ventricle but also of the left ; the impediment at the right side to the return of the blood reacting upon the arterial capillaries of the general system, and backwards upon the arteries and left ventricle, the increased efforts of which lead to hypertrophy. In the majority of cases, however, the systemic capillaries relieve themselves by allowing the more watery parts of the blood to transude, and anasarca of the extremities rather than hypertrophy of the left ventricle is the result.

Certain trades or occupations which require the body to be maintained for a long time in a constrained or unnatural position,

\* The Lancet.

such as that of tailor, shoemaker, &c., may likewise, by impeding the descent of the diaphragm, and thus interfering with the free movements of the heart, indirectly bring about disease of the organ.

When the heart's action is frequently, and for a length of time, accelerated by inordinate muscular exertion, this may lay the foundation for disease; increased action leading to increased nutrition, and increased nutrition to increased development. Under such circumstances, if the subject is young and healthy, hypertrophy of the muscular parietes of the heart may ensue. On the other hand, if the muscular fibre is lax, and the individual is debilitated by previous illness or other cause, the walls of the ventricles instead of increasing in thickness, may yield to the distending force from within, and their cavities eventually become dilated. M. Flögel\* thinks that among the determining causes of cardiac disease, immoderate, long-continued, or even only momentary bodily efforts, especially of the muscles of respiration, of such kind as interfere with the free performance of respiration, have not received the attention their importance deserves. He gives five cases in which the patients referred their cardiac symptoms to muscular efforts. Dr. Taylor, on the other hand, considers doubtful if either unusual muscular efforts, or habits of intemperance ever cause hypertrophy.

Advanced age would appear to be a predisposing cause of cardiac disease; we seldom, in examining the bodies of aged subjects, find the endocardium covering the valves in a perfect normal condition, this tissue would appear to undergo a change as life advances (for the same reason that the prostate gland enlarges it becomes opaque, loses something of its elasticity, and eventually becomes, often, the seat of atheromatous or calcareous deposits). This frequently occasions little or no disturbance of the heart's functions; it becomes a source of danger, however, if inflammation of other organs supervenes, particularly of the lungs or bronchial mucous membrane.

Corvisart looked upon protracted mental emotion, whether of an exciting or depressing nature, as a powerful predisposing cause of cardiac disease. It must be recollected, however, that Corvisart's remarks are founded upon cases which came under his

\* Brit. and For. Med. Rev., 1845.

observation during, and subsequent to the first French revolution; indeed he alludes to this very circumstance himself, in the chapter upon the causes of cardiac disease. "The scenes of the revolution, and the dreadful results which succeeded—the sudden reverses of fortune—the grief, the misery, and the violent emotions occasioned thereby, have recently furnished numerous proofs of the influence of mental causes in the development of organic disease, and of the heart in particular." "How frequently have we had in hospital individuals who were formerly in opulence, now reduced to the lowest state of poverty, who anxiously looked for death as a termination of their misery, which disease of the heart brought less rapidly than they desired."

#### PROGRESS AND DURATION OF CARDIAC DISEASE.

It is a common opinion, and not limited to non-professional individuals, that disease of the heart once established must terminate fatally; and a person whose case is pronounced to be such, is impressed with the conviction, that it must have a fatal and perhaps a sudden termination. This opinion has received confirmation from the writings of medical men themselves, and the well-known motto to Corvisart's work, "*hæret lateri lethalis arundo*," has been often quoted in support of it. Since the discovery of auscultation, however, our means of diagnosis have been considerably improved, the distinction between the several forms of cardiac disease has been laid down with much greater precision, and it does not admit of doubt, that, many morbid conditions of this organ have not the tendency to run on to a fatal termination that was formerly supposed; and, many of them likewise may be kept in check by appropriate remedial measures.

"Confirmed organic heart-disease, absolutely insusceptible of cure as it is, carries with it," Dr. Chevers\* observes, "the redeeming point, that, in its ordinary forms, the victim's life is allowed a more lengthened respite than is the case in any other description of destructive chronic disease with which we are acquainted—a respite which, it is true, is fraught with some amount of suffering, great need of self-denial, and many perils; but during which, if the self-denial be rigorously maintained, while the sources of peril are carefully avoided, the suffering may

\* Treatise on Diseases of the Heart, Calcutta, 1851.

be in a great measure mitigated, and the term of existence be prolonged to a degree, the full limits of which are probably far more extended than we are at present aware of. We know that the most ordinary forms of exocardial and endocardial disease—adherent pericardium and valvular contraction, certainly do not generally and of necessity, destroy life by their own advance, and by the influence which they exert in gradually impairing other organs, in less than from ten to fifteen years; and the duration of their progress is found to be occasionally longer even than this.”

The rapidity or the slowness of the progress of cardiac disease is influenced by a variety of circumstances, such as the seat, the nature, and the form of disease under which the patient labours; its acute or chronic character, and whether it is simple or complicated; its stage, and whether it has, or has not been succeeded by secondary lesions of the heart itself, or of some other important organ; whether it is or is not associated with a morbid condition of the blood, or with a cachectic state of the general system. The age of the patient will also have some influence on his habits, whether intemperate or the contrary; his occupation or trade, and particularly his station in life, whether it places him above the necessity of daily toil, or whether he is obliged to labour for his bread, exposed perhaps to every vicissitude of the weather, with perhaps an insufficient supply of food, or scanty clothing.

The acute inflammatory affections of the heart are the most rapid in their progress, and they may either end in resolution, or terminate fatally in the acute stage, or pass into the chronic form. Cardiac disease is, however, seldom fatal in the acute stage, unless some serious complication exists; or unless the disease arises in a broken down constitution. If, however, the inflammation passes certain limits, although the patient recovers, organic changes ensue which are permanent, and will necessarily tend to shorten life.

The duration of the chronic forms of cardiac disease is very variable; as a general rule, almost, their progress is slow; and although, when once established, organic disease of the heart is incurable, we can in most cases mitigate some of its ill effects; we can prevent congestion, or remove it when it occurs; and under favourable circumstances, we are able in many instances to ward off dropsy, and to prolong life.

The progress of the chronic forms of organic disease of the heart is by no means uniform ; in many, indeed in most instances, there are remissions, sometimes amounting almost to intermissions of the symptoms, lasting for a longer or shorter period, and occurring at variable intervals. This is not peculiar to disease of the heart, for the same thing is not unfrequent in aneurism of the arch of the aorta : in some cases of the latter, there are periods during which, if auscultation were not employed, the disease would appear to be almost latent. The remissions in organic disease of the heart are often, of course, the result of treatment, but they occur independent of any ; indeed sometimes when the patient had violated all dietetic, or other rules laid down for his guidance.

The period of remission in these cases is termed, by Dr. Ritchie\* "the period of latency or quiescence ; it may remain undisturbed," he observes, "in favourable circumstances, if the case be aortic, for so long a time as 15 or 20 years." This, however, must be very rare. "It happens chiefly," he adds, "in persons not exposed to causes which disturb the circulation, increase the quantity of blood, or diminish greatly the vital energy." The original cause of the exacerbation is generally some indiscretion on the part of the patient, as intemperance, exposure to cold, fatigue, damp, over-exertion, too free living, or strong mental emotion, &c., and its immediate cause is generally congestion of the lungs, or bronchial mucous membrane, of the liver, or other abdominal viscera.

The seat and nature of the cardiac lesion, as well as its extent or amount, have considerable influence in this respect ; thus a diseased state of the aortic valves, permitting regurgitation, is a more formidable lesion than a state of the mitral orifice permitting regurgitation ; and it is scarcely necessary to say that a very contracted condition of either of the orifices at the left side of the heart, is a much more serious lesion, than such an amount of disease as does not materially interfere with the circulation through the organ.

Again, the cause which has given rise to the disease is not without its influence ; thus, a valvular lesion, the result of inflammation, is generally a more serious affection than a morbid state of the valve, the result, simply, of adventitious deposit in advanced

\* Edinburgh Medical and Surgical Journal, No. 185.



life ; owing to the subsequent shrinking and contraction which takes place in the exudation poured out, as the result of inflammation ; by which the orifices are further diminished in size, or the valves are rendered less capable of fulfilling their function. On the other hand, in subjects advanced in life, who have died of various diseases, atheromatous or calcareous deposit is frequently found upon the valves at the left side of the heart, although the patient during life had never exhibited any symptom leading to suspicion of its presence.

Hypertrophy of the muscular tissue of the ventricles, is almost always secondary to some other affection of the heart, of the great vessels, or the lungs ; when an impediment has existed for a longer or shorter time to the passage of the blood through, or out of the heart. When moderate in degree, and when uncomplicated with disease of other parts, hypertrophy is not the formidable affection it was once supposed, and a person, the subject of it, may enjoy good health, and may attain advanced age, provided his habits are temperate, and he is not exposed to those disturbing influences which tend to deteriorate the general health. Indeed, hypertrophy instead of being regarded as a form of disease which requires remedies directed towards its removal, is now, very properly looked upon as a compensatory process, and nature's remedy for overcoming obstruction, guarding against over distention of the cavities, and preventing dilatation. Dr. Ridge, and Dr. Chever have strongly advocated this view ; " I have not seen or heard any case," Dr. Chevers\* observes, " in which it was demonstrable that excessive muscular development, and strengthening of the walls of the heart existed, otherwise than as a compensation for an impediment of some kind, or for the weakening attendant upon temporary or permanent dilatation of its cavities ; nor have I been able to meet with a heart in which the amount of hypertrophy appeared to be more than compensatory for the coincident dilatation or impediment : whereas, in nearly every complete case the symptoms during life, as well as the sum of the morbid appearances, rendered it almost a matter of demonstration, that the amount of hypertrophy was not, and had never been, fully sufficient to counteract the impediment or embarrassment to which the organ was subject.

\* On Diseases of the Heart.



Some dilatation of the cavity of the ventricles, invariably, accompanies hypertrophy of the muscular walls of the heart; when the dilatation, however, much preponderates over the hypertrophy, but particularly when dilatation is combined with attenuation of the parietes of the left ventricle, the prognosis is always more unfavourable, and the progress of the disease is likewise more rapid. If, in addition, the muscular tissue of the heart is softened, or has undergone fatty degeneration, or fat has taken the place of the muscular fibre, we can do little more than palliate symptoms, and endeavour to prolong life. The prognosis, it is scarcely necessary to say, is exceedingly unfavourable, and death is frequently sudden.

The occupation of the patient, and the kind of life which he leads, have also an influence upon the rapidity of the progress of organic disease of the heart. If the patient's trade is laborious; if it is one in which he is constantly exposed to the vicissitudes of the weather; if, in addition, he is addicted to intemperance and excesses, and if, at the same time, he is badly fed, badly clothed, and badly lodged, the disease, whatever it is, will run its course rapidly, and treatment will have little effect. "The man," as Dr. Latham says, "who having an unsound heart, must traffic with his sinews for his daily bread, has a poor chance of benefit from medicine."

Age exerts a decided influence upon the duration of cardiac disease; thus, congenital lesions or malformations often prove fatal soon after birth; and Corvisart has made the remark, which experience fully bears out, that cardiac disease, particularly valvular disease developed in infancy, generally terminates fatally, from the twelfth to the sixteenth year. Indeed, endocarditis, in very young subjects, not only runs a more rapid course than in the adult, but, if the patient survives the attack, the heart remains seriously involved, and a train of phenomena follow, which are highly characteristic, of which we occasionally have the opportunity of seeing examples in hospital; I allude to a defective development of the muscular and osseous systems, and an apparent cessation of growth, dating from some period subsequent to the invasion of the heart disease. It is generally observed in individuals, who, when young, had laboured under pericarditis and endocarditis, terminating in valvular disease; and the affection can be often traced

to an attack of acute rheumatism, though it is also of individuals who have never suffered from the latter disease.

If the patient is a female, the mammæ at the age of puberty are scarcely developed, the catamenia do not appear, the pelvis resembles that of a child, and the breasts have not the rounded form which they acquire in health at this age. If the patient is a male, the signs of approaching puberty are hardly evident; the genital organs are but scarcely developed; the beard scarcely shows itself; the bones are small, the muscles small and flabby. In both sexes the countenance acquires an anxious and sorrowful expression; the stature is small, the thorax narrow, and often malformed, the left side protruding outwards in the præcordial region, the ribs being protruded on this side predominating over the right, with, often, lateral curvature of the spine. In some cases the mind even appears to be affected by the want of physical development of the body. "Notwithstanding the functions being performed with vigour," robust health is often enjoyed. Indeed it is easy to conceive (as Dr. Willis says) "that vigorous health of the body at large, is incompatible with a failure of function in the organ, upon which all parts of the system depend immediately for their supplies of nutriment."

We have had several patients in hospital who verified these observations completely, and who, though approaching old age, appeared from their size not to be more than nine or ten years of age. In the majority of these cases, the cardiac affection was distinctly traced to an attack of acute rheumatism; in the remainder, to pericarditis or endocarditis subsequent to exposure to cold, or following an injury of the left side of the chest.

#### TERMINATION OF CARDIAC DISEASE.

Disease of the entire heart is seldom or never met with. A morbid alteration may originate in, and be limited to the endocardium, the membrane, to the lining membrane, or to the muscular wall of the heart, or to a small portion only of one of these tissues. The two sides of the heart scarcely ever suffer in an equal degree, for while morbid conditions of the valves at the left side of the organ are extremely common, disease of the same parts of the right side is extremely rare.

Organic disease of the heart more frequently owes its fatal termination to its complications, and to the secondary lesions which result from it, than to direct failure of the heart's action. Indeed, "this organ," as Dr. Chevers remarks in the valuable work from which I have before quoted, "is endowed with an almost endless variety of beautiful compensatory resources, and means of adapting itself to circumstances of temporary embarrassment, and of fixed disease, so that no shock which falls short of absolutely suspending its nervous supply, nor any injury which is less than sufficient to destroy some vitally essential portion of its mechanism, has the power at once to disable it wholly."

The termination of cardiac disease is sometimes sudden; in the majority of cases, however, it is gradual, and the patient dies after a more or less protracted struggle; the immediate result being hastened by pulmonary or bronchial congestion, or inflammation, by effusion into the pleural cavities, or by the formation of fibrinous concretions in the cavities of the heart.

It were a sad story, as Dr. Ormerod,\* in his admirable lectures observes, to tell how patients with disease of the heart die; the tragedies, so to say, of the medical wards of our large hospitals; "How some wrung with pain, have struggled in the week-long agony of death. How some have, for days together, fixed themselves in the most fantastic postures, the only way in which they could find relief; some leaning forwards, resting their heads on a stool to catch a few minutes' sleep; some on their hands and knees, till the approach of death, blunting their sensations, allowed them to lie down—a sure sign of coming dissolution." The patient usually preserves his senses perfectly to the close, though when a considerable impediment to the pulmonary circulation existed, and the venous blood has been prevented from undergoing the necessary changes in the lungs, death is occasionally preceded by stupor, or by a mild kind of delirium.

When the death is sudden, it may be the result of rupture of the parietes of the heart, or of cerebral or pulmonary apoplexy, or syncope may supervene, from which the patient never rallies; or on some slight exertion on the patient's part, the over distended and weakened left ventricle becomes incapable of contracting upon

\* Gulstonian Lectures—Medical Gazette.

its contents, the coronary circulation is impeded or interrupted and the heart's action necessarily ceases.

The immediate cause of *sudden* death in the majority of the cases, is, probably, impediment to the coronary circulation; and when we come to examine such cases *post mortem*, we are sometimes surprised, rather that the heart should have continued so long to perform its functions, than that it should have ceased to act at that particular moment.

## CHAPTER X.

OF DISEASES OF THE HEART—DISEASES OF THE INVESTING  
TYPES OF PERICARDITIS—ANATOMICAL CHARACTERS OF PE-  
SYMPTOMS OF PERICARDITIS—CONSTITUTIONAL AND LOCAL  
PHYSICAL SIGNS.

the anatomy of the heart, we said that several distinct into its composition; thus the principal part of its up of muscular fibre; this is invested externally by rane, and lined internally by a membrane having r with that lining the larger vessels; while fibrous into the composition of certain parts of the valves, and e capsule of the heart; and the whole organ is con- ro-serous sac.

ral rule, disease originates in a single tissue, to which ited; and a simple classification admits of being made umstance. It is true that when disease has persisted one tissue, morbid alteration of another often ensues; ractical purposes, an arrangement founded upon the of the disease appears to be the most convenient.

ent of any structural change, the functions of the deranged or disturbed in various ways, constituting ned the inorganic affections of the organ. Hence ditions of the heart admit of being sub-divided into sses, viz.: Organic diseases; and Inorganic affec-

unic Diseases of the heart will be described in the er:

es of the investing membrane; under which pericar- ffects will be considered.

es of the lining membrane; which will include endo- ll the varieties of valvular disease.

es of the muscular tissue; under which myocarditis,

dilatation, and hypertrophy, fatty disease and softening, rupture of the heart, and cardiac aneurism will be described.

#### DISEASES OF THE INVESTING MEMBRANE OF THE HEART—PERICARDITIS

The morbid changes met with in the investing membrane of the heart, are, in the great majority of cases, the result of inflammation. Acute inflammation of this membrane is sufficiently common, and presents generally positive and characteristic signs; yet, until within a few years, its diagnosis was looked upon as one of the most difficult problems in pathology; the disease was rather guessed at than diagnosed during life, or it was mistaken for inflammation of some other organ, or entirely overlooked. "So vague and irregular are its symptoms, and so proteous-like are its forms (Mr. Burns\* observes), that were a patient with this affection presented to a practitioner, who had not had several opportunities of witnessing its progress, and seeing the morbid appearances, he would, from the attendant symptoms, suspect any other organ sooner than the heart, and the presence of any other morbid condition rather than inflammation or any of its modifications." Ever up to the time at which Laennec† wrote, little advance seems to have been made in this respect. "There are few diseases (he observes) more difficult of diagnosis than pericarditis. I have sometimes seen its existence conjectured; I have myself done so, for I do not think the term *recognise* can be used when there are no certain signs of its presence." M. Louis,‡ writing also in the year 1826, observes, in reference to the same point, the "obscurity of the diagnosis of pericarditis is still such that it would be perhaps impossible to quote an exact and certain history of this affection terminating in a return to health."

Since the comparatively recent period at which Laennec and Louis wrote, cardiac pathology has, however, made considerable and important advances; and the obscurity which long hung over the diagnosis of pericarditis, has been, in a great measure, dispelled so that, at the present day, it is as easily recognised as inflammation of any internal organ, and it is perhaps diagnosed with greater facility than inflammation of any other tissue hidden from our view.

*Types of Pericarditis.*—Inflammation of the pericardium m

\* On Diseases of the Heart.

† Ausc. Med. Paris: 1826.

‡ Mem. Anat. Path. Paris: 1826.

as an idiopathic affection, which is rare, or it may arise in the progress of acute rheumatism, or some other morbid condition, which is sufficiently common. It may assume either a sthenic or asthenic type: it may run either an acute or a chronic course. The inflammation may engage both surfaces of the serous membrane, and extend to the fibrous layer, or to the subjacent cellular fibres; or it may be partial, and limited to the serous membrane investing the heart or lining the sac, or to only a portion of the heart. Finally, it may be complicated with inflammation of the lungs, or pleura; with carditis, or endocarditis; with chronic inflammation of the valves, or morbid conditions of the muscular tissue of the heart; with bronchitis, or emphysema of the lungs. Hence it is not surprising that its symptoms should exhibit considerable variety, or that its prominent features should vary remarkably in different subjects.

For instance, pericarditis may, as I have said, assume either a sthenic or asthenic type, and whether it assumes the one or the other will depend, not alone upon the causes which have called the disease into existence, but upon the healthy or unhealthy state of the patient's constitution, and upon the normal or abnormal condition of his blood at the period of its invasion. Thus, in a sthenic and vigorous subject, and when the disease supervenes in the course of acute rheumatism, all the signs of the sthenic type of inflammation will be well marked, the fever will be purely inflammatory, the blood, containing a large amount of fibrine, coagulates readily, and exhibits the buffy coat well marked, and the inflammatory exudation consists of plastic lymph, which has a strong tendency to become organized, and from this circumstance has been termed "*euplastic*."

On the other hand, when pericarditis occurs in a subject whose constitution is broken down, whose constitution is debilitated by previous disease, or other cause, and whose blood is impoverished or detested, the fever will assume a different type, and the inflammation will be of the *asthenic* character; the blood coagulates imperfectly when drawn, and serum, rather than lymph, will be effused into the pericardial sac, or at least will greatly preponderate. The lymph effused under such circumstances has little tendency to become organized, and from this circumstance has been termed "*cacoplastic*."

In still lower forms of inflammation, and where the blood is more seriously deteriorated, "more or less of the product of inflammation is, as Dr. Williams\* observes, *aplastic*, and totally incapable of organization, and is thrown off with the liquid in separate large globules constituting pus, or in detached flakes or curds." Under such circumstances the pericardical effusion consists of a serous or sero-purulent fluid, mixed with flakes or fragments of lymph.

#### ANATOMICAL CHARACTERS OF ACUTE PERICARDITIS.

The serous surfaces of the pericardium when inflamed, undergo similar pathological changes to those which occur in other serous membranes; but owing to the constant motions of the heart, the appearances presented are somewhat peculiar in this membrane.

*Increased Vascularity.*—The earliest effect of inflammation upon the pericardium is said to be the suspension of the natural lubrication, which in the normal state lubricates this membrane, and permits of its opposed surfaces gliding over one another without producing friction; the effect of which would be to render the surfaces drier than natural, and to give rise, as in the pleura and peritonæum, to a slight grazing sound on auscultation. We have, however, no positive evidence that such ever occurs here. Taylor, it is true, mentions an instance where "the pericardium externally was dry and transparent, like oiled silk," but the patient did not die at this early period of the disease. An early symptom mentioned by French writers is a sticky feel of the membrane such as is given by fish some hours out of the water, which is named *poissonneux*, from this circumstance. It, however, more properly belongs to the next stage, that of exudation. The early changes are diminution in transparency and injection of the membrane; the latter may consist of dots or patches of a red color formed by a network of capillary vessels seated in the sub-serous tissue, or "the serous surface of the pericardium itself assumes the appearance of soft red velvet, having obliquely erect piles, looking pale and turbid, as if infiltrated."† We seldom, however, have the opportunity of examining the morbid appearances in pericarditis at this early period.

*Exudation Stage.*—The next effect of inflammation is the

\* Elements of Medicine.

† Rokitansky, Man. of Pathol., vol. iv.



udation of a plastic coagulable matter, or of a matter which is not coagulable; the former adheres to the surface of the membrane, the latter distends the pericardial sac; and this may occur within twenty-four hours from the commencement of the attack. When the inflammation is acute, occurs in a healthy subject, and the type of the fever is sthenic, the exudation as a general rule consists principally of plastic or coagulable lymph. When the inflammation occurs in an unhealthy, broken-down subject, the type of the fever is asthenic, and the exudation consists principally of non-coagulable matters—viz., serum mixed with lymph, more rarely with pus, or with the red particles of the blood. The former is sometimes termed the *dry* form of pericarditis, the latter the *liquid* variety. The two kinds of exudation may, however, occur simultaneously, and the symptoms may be referrible at first to the one, and subsequently to the other.

*Exudation of Lymph.*—The lymph effused is a soft, semi-transparent, jelly-like matter, which adheres to the free surfaces of the pericardium, and forms at first a thin, almost diaphanous film, which has been compared to hoar frost, or to a layer of liquid gelatine spread upon the parts with a camel's hair pencil. It is generally deposited upon the serous layer investing the heart before it engages the layer of this membrane which lines the pericardial sac; it may be partial and circumscribed, and engage only a portion of the membrane, particularly that part which covers the base of the heart and the origin of the large vessels; or it may be general, and in larger amount, and cover the entire of both surfaces of the pericardium; or it may be loosely interposed between the two layers of the membrane. Its consistence is about that of jelly; its colour varies, being sometimes yellowish or grayish, at others of a reddish or brownish hue.

This lymph being soft and adhesive, becomes altered in appearance, owing to the constant friction to which it is exposed during the movements of the heart. Thus in some cases it is beautifully reticulated, resembling lace work, at others it resembles the section of a piece of sponge, or the interior of the gall-bladder, or the honeycomb stomach of ruminant animals, to which Corvisart compared it. In others it presents a granular, papillary, or villous appearance, compared by Laennec to that produced by separating two slabs coated with butter or grease, or more nearly resembling

the surface of the tongue of some of the feline carnivora. others, again, it is shreddy and "flocculent like tow;" the latter appearance is, however, in general, partial. In more chronic cases it occasionally presents a remarkable mammillated appearance resembling the exterior of a pine-apple, or the botryoidal form of some minerals. Finally, Dr. Hope mentions a case where it was arranged in transverse plaited wrinkles, resembling upon a small scale the undulations of sand upon the sea-shore. The peculiar appearance of the false membrane in the latter cases is the result of the deposit of successive layers of lymph, owing to the repeated recurrence of the inflammation, sometimes to its long continuance in a chronic form.

*Exudation of Liquid.*—When the inflammatory exudation is principally or wholly liquid, this consists of a serous fluid more or less opaque and turbid, mixed with flakes or fragments of lymph and resembling whey, or very thin Indian-meal gruel, to which Dr. Swett compares it. Sometimes it is slightly tinged, at other times deeply coloured with the red particles of the blood, constituting what Laennec termed hæmorrhagic pericarditis; this, however, is in general the result of secondary exudation. If the inflammation persists long, or if the fever is of a lower type, the exudation may have a sero-purulent character, or may consist principally of pus. The latter, however, is only observed in the more chronic form or when there have been successive renewals of the inflammation or the patient is of a broken-down constitution. When the exudation consists of liquid, whatever be its nature, the opposed surfaces of the pericardium will be kept asunder by it; while if it is large in amount, the heart's movements will be impeded, and the organ sometimes injuriously compressed.

The rapidity with which exudation occurs is sometimes remarkable; within less than twenty-four hours we may find a considerable amount of fluid; in general, however, it takes place more slowly and gradually, and from two to four days elapse before much is effused. The effusion may (Dr. Law observes) exist for only twenty-four hours, when absorption takes place, and may reappear again within a short period. The amount varies from a few ounces to several pints; of thirty-six cases mentioned by Louis, the amount of fluid was very small in three only; in a few, from eight to ten ounces; and in the remainder it varied

from one pint to four. Corvisart mentions cases where the amount much exceeded this, in one reaching to eight pounds. Dr. Swett\* likewise gives one where eight pints of liquid were contained in the pericardial sac. The pericardium in this case occupied the whole anterior part of the chest, the diaphragm was protruded downwards, and formed a very large convexity towards the cavity of the abdomen; the liver was also protruded downwards, and both lungs were pushed into the posterior and lateral regions of the thorax. In the hæmorrhagic form of pericarditis described by MM. Seidlitz and Kyber, from eight to ten pints of liquid exudation are said to have been by no means uncommon. Under favourable circumstances, the liquid may be entirely absorbed, when the opposed surfaces of the pericardium come into contact, and adhesion may take place between them. This is a result which cannot, however, always be attained, the patient sinks before it occurs, or other changes ensue.

*Stage of Adhesion.*—When the inflammatory exudation consists principally of lymph, or of both lymph and fluid; after the absorption of the latter, the opposed surfaces of the membrane coated with lymph come in contact, adhesion more or less close takes place between them, over a greater or less extent of surface, according to circumstances. Even when lymph has been effused upon only one surface of the membrane, still adhesion may take place, in consequence of “inflammation from contact” being set up in the opposite surface. Dr. Copland† has shown that in *peritonitis*, the lymph effused “rapidly irritates and inflames the opposite surface, or that coming in contact with it;” and analogy would lead us to expect the same to occur in pericarditis. The period which intervenes between the development of pericarditis and the stage of adhesion varies necessarily according to the nature of the exudation; in the sthenic form of the disease it may be completed within a few days; indeed, in very young subjects, the two layers have been found to be adherent on the third day; the earliest period at which I have found it to occur in the adult was the eighth day. The adhesions may be either general or partial; when partial, they may consist simply in a few bands which pass across from one serous surface to the other, or hang loosely from one, without any adhesion to the other; the result either of a par-

\* On Diseases of the Chest.

† Dict. of Medicine.

tial deposit of lymph, or of the parts having been kept asunder some time by liquid exudation. This is most commonly observed at the apex and about the origin of the large vessels. The adhesions, in the first instance, are slight and easily broken up, and opposed surfaces of the pericardium can be readily separated from one another; eventually the plastic lymph is converted into cellular or dense cellulo-fibrous tissue, and the union becomes so close that the two layers of the pericardium appear to constitute but a single membrane; when (if the adhesions are general) the cavity of the pericardial sac is completely obliterated. This appears to have been the morbid state which was occasionally mistaken by the earlier pathologists for congenital deficiency of the pericardium.

When inflammation of the pleura is combined with pericarditis, lymph is likewise effused upon the outer surface of the pericardial sac, and adhesion takes place between the pericardium and pleuræ, and the tissues immediately around; and when we come to examine the parts, it appears just "as if a quantity of liquid size had been poured in, so as to fill up every interstice, and then become organized."\*

#### ANATOMICAL CHARACTERS OF CHRONIC PERICARDITIS.

Acute inflammation of the pericardium generally runs its course within two or three weeks; if it persists longer, it may be set down as having passed into the chronic stage. It sometimes happens that when a patient is convalescent from an acute attack, a relapse occurs, and several recurrent attacks may follow; occasionally inflammation is subacute or chronic from the outset. The exudation under such circumstances always consists of a certain amount of non-organizable matter, principally liquid, which is usually abundant, because "the inflammation is continually adding to the amount on the one hand, and preventing its absorption on the other." The liquid consists of a turbid serum mixed with fibrin of lymph or with pus, and as the one or the other predominates it will have a serous or sero-purulent character; in a few instances it consisted principally of pus. The surfaces of the pericardium are usually at the same time coated with false membrane, which varies in thickness in different cases, but is always more consi-

\* Dr. Macleod on Rheumatism.

able than in the acute form of the disease. Sometimes, in consequence of partial adhesions, the liquid appears to be contained in distinct cells; in others, bands or shreds of lymph of various lengths connect the opposed surfaces; or the latter are attached by one extremity, the other floating loosely in the cavity.

It occasionally happens, as has been said, that when a patient is recovering from acute pericarditis, the inflammation returns, though in a less intense degree, and this may occur several times; each recurring attack is accompanied by fresh exudation, and the latest effused being less susceptible of organization than the preceding, adhesion does not take place, and the auscultatory signs of pericarditis may be present for a considerable time. More frequently, however, adhesion is prevented by the liquid exudation which distends the pericardial sac. Ultimately the false membrane in these cases acquires a remarkable thickness, as much as from two to twelve lines; it assumes a fleshy appearance and a fibrous structure, occasionally presenting the botryoidal or mammillated appearance previously mentioned; sometimes it has a dark red hue, being stained by the colouring matter of the blood.

Again, the exudation deposited by one access of inflammation may itself become (as Rokitansky observes) the site of a new inflammation, during the time that it is becoming, but is not completely, organized. "This secondary inflammatory process deposits exudation both upon the free surface of that product, and also within its tissue or parenchyma: the former constitutes a second free exudation; the latter an infiltration. The infiltrated product during its organization becomes an integral part of the original exudation, renders it thick and very dense, compact in its parenchyma, and of fibroid or fibro-cartilaginiform structure." Hæmorrhage occasionally accompanies these secondary exudations. If the patient recover, the fluid is absorbed, adhesion takes place between the two laminae, and eventually "the heart is enclosed in a thick, tough, unyielding casing."

*Osseous Concretions.*—The fibroid exudation occasionally becomes the seat of osseous concretion, phosphate of lime being deposited in plates or lamina of variable size. "Osseous concretions are scarcely ever developed (Rokitansky observes) except in the fibroid exudation after the lamellæ have become fused together, and the pericardium has been thus made to adhere to the heart by

this dense and resisting fibroid medium." The osseous deposit presents itself in the form of plates or laminæ of different shapes and sizes; in some rare cases forming a kind of ring round the body of the ventricles; this, according to Meckel, is an effort of Nature "to convert a membranous into an osseous cavity, similar to what prevails in the cranial and vertebral cavities." The muscular tissue of the heart, under such circumstances, generally undergoes degeneration; its tissue becomes softer and more friable, and its colour paler.

#### TUBERCULAR PERICARDITIS.

A tuberculous state of the blood (Mr. Ancell observes\*) is known to operate as a predisposing cause of inflammation of serous membranes, with specific symptoms; tubercles are, however, rare in the pericardium, in comparison with the peritoneum or pleura. The first writer who gave an accurate description of tubercular pericarditis was Dr. G. Burrows;† Dr. H. Kennedy likewise has called attention to it;‡ the morbid appearances have also been described by Hasse, Rokitansky, and others.

Some difference of opinion exists among pathologists as to whether the tubercular deposit is primary or secondary; in other words, whether the tubercles are to be regarded as the cause of the pericarditis, or as simply a product of the change which the inflammatory exudation undergoes. According to Dr. G. Burrows, "The tubercles are deposited in the first instance as a result of tubercular cachexia; they then excite inflammation of the serous membrane; fresh crops of tubercles springing from the same state of constitution, keep up the inflammatory action, and thus the acute is converted into chronic pericarditis." According to Laennec, Hasse, and Rokitansky, tuberculosis rarely manifests itself in the pericardium in any other form than as a product of inflammation.

The tubercular variety of pericarditis is essentially chronic; the inflammatory exudation in it is usually abundant, and consists always of a considerable proportion of liquid, analogous to that effused in other serous membranes as the result of inflammation in such constitutions. "The tubercles seldom go on to complete softening, because death, when it ensues, is generally occasioned

\* A Treatise on Tuberculosis, 1852.    † Med. Chir. Trans., vol. xxx., 1847.  
‡ Dub. Med. Press, Jan., 1853.

by the pericarditis, or the subsequent tuberculous secretions, or by general cachexia;" and in such cases tubercles of old or recent date are almost invariably found in other situations in addition, as the lungs, bronchial glands, the peritoneum, or the pleura; from the latter, indeed, they may extend to the pericardium.

Tuberculosis of the pericardium is rare in the adult, but more common in early life; MM. Rillett and Barthez state that they found tubercles in the pericardium ten times in 312 young subjects.

## HÆMORRHAGIC PERICARDITIS.

The secondary inflammatory exudation of pericarditis has sometimes a deep red hue, occasionally the dark colour of coffee grounds, and consists in part of blood, or it is deeply stained by the colouring principle of the blood, constituting the hæmorrhagic form of pericarditis of Laennec. According to Rokitansky, this is, in general, the result of a secondary inflammatory process occurring in a plastic exudation. "The structure in which the inflammation occurs is in course of organization, its vessels are only just forming, and have as yet no actual coats, or at any rate but very delicate and permeable ones, and they have not yet united into a freely inoculating circulatory system; from such a structure the exudation occurs repeatedly and at intervals." "The whole process bears throughout it the stamp of an inflammation which has not arrived at maturity, and its product is blood, altered by congestion in the composition, and mutual relation and intermixture of its elements."

A form of pericarditis, however, to which the name hæmorrhagic more properly belongs, has been described by M. Seidlitz\* of St. Petersburg, under the name "*Pericarditis exsudatoria sanguinolenta*," and by M. Kyber† under the name "*Pericarditis scorbutica*." The affection was observed by M. Seidlitz principally among sailors at St. Petersburg, at the period of the year when scorbutic affections ordinarily prevail; and according to M. Kyber, it is frequent on the extreme northern coasts of Europe where scurvy is endemic. In sixty subjects who died of scurvy, M. Karawagen‡ found that thirty had this kind of pericarditis; the fluid was dark and amounted to four or five pints. It occurs either

\* Brit. and For. Med. Review.

† Ed. Month. Jour., Mar. 1840.

‡ Medicinische Zeitung Dec., 1840, and Brit. and For. Med. Rev., vol. xii.



in an acute or chronic form, the former being commonly primary, the latter a secondary affection supervening upon catarrhal or rheumatic disease.

The following were the morbid appearances observed by M Kyber: "The pericardium enormously distended, often measuring a foot in length, and containing three to eight or even ten pounds of dark red, or blackish opaque fluid, composed of serum or fibrin, with blood globules altered in form. Inner surface of the pericardium covered with a coat of lymph reticulated on the free surface, and of the colour of cinnamon; the membrane itself either injected or stained of a dark colour. On the heart, the lymph often irregularly disposed in shreds, having a rugged or honey-comb appearance. The heart diminished in size, its substance pale, flaccid, and easily torn; the ventricles greatly compressed and empty; the left lung compressed and bloodless. In cases where the liquid had been absorbed, adhesions were found between the layers of the pericardium."

The symptoms of the acute form were as follows. It commenced with "a sensation of coldness and prostration, oppression, alternating with pains in the chest and epigastrium, rapid painless breathing, without cough, and decubitus on the left side; succeeded by a discontented gloomy condition, or complete apathy, with the pulse small, intermittent, or when the effused fluid reaches two or three pounds, inappreciable. When the quantity of fluid is very large the extremities are cold, the countenance bloated, the pupils dilated, the jugular veins distended, the expression exceedingly anxious, prostration of strength, but consciousness remains unaffected. The sound on percussion may be dull on the left front up to the clavicle; heart's sounds faint and remote; inaudible if the fluid be large in amount; if otherwise there may be friction sound, heart's impulse imperceptible. The left side of the thorax is distended, and does not move freely; the lung on this side does not act, the right side on the contrary has puerile respiration. Epigastrium protruded and sensitive on pressure; pain referred likewise to the anterior surface of the chest, and to the right hypochondrium. These symptoms may be developed in twelve hours, and death usually takes place suddenly."



## SYMPTOMS OF PERICARDITIS.

The symptoms of pericarditis for the convenience of description may be considered under three separate heads—viz :

1. The general or constitutional symptoms.
2. The local symptoms.
3. The physical signs.

The most remarkable of the general or constitutional symptoms, and those only which it will be necessary to notice individually, are—1, inflammatory fever; 2, the condition of the pulse; 3, the expression of countenance; and 4, the position which the patient assumes in bed.

The local symptoms from which most information is to be derived are—1, palpitation; 2, pain; and 3, dyspnoea.

The physical signs are those afforded by—1, inspection of the chest; 2, palpation, or the application of the hand; 3, percussion; and 4, auscultation.

## GENERAL OR CONSTITUTIONAL SYMPTOMS.

*Inflammatory Fever.*—Acute inflammation of the pericardium, equally as of other serous membranes, is accompanied by symptomatic fever; the information, however, to be derived from this sign is not always of much value, because in the majority of cases the disease arises during the course of acute rheumatism, which itself is attended with a high degree of fever; and in the comparatively few instances in which we meet with pericarditis as an *idiopathic* affection, the inflammation is generally not limited to the pericardium, but is combined with inflammation of the pleura or lungs, or with both. The intensity of the febrile symptoms in pericarditis, as in other inflammatory affections, is generally in proportion to the acute character and the extent of the local inflammation. Thus, when the inflammation is acute and its type sthenic, when much of the pericardium is engaged, and the patient is otherwise healthy, all the signs of inflammatory fever will be well marked, the exudation will consist of plastic lymph, and the physical signs will be those of the dry form of pericarditis. This is its type in the great majority of cases of rheumatic pericarditis. On the other hand, when pericarditis occurs as an idiopathic affection, or as a sequela of small-pox, scarlatina, &c., or when it arises in the course of diffuse inflammation, Bright's disease of the kidney, &c., the type of the inflammation, as a general rule, is asthenic, or if it had

been sthenic at first, it soon changes, and the inflammatory exudation consists principally of liquid, which, in broken-down constitutions, may assume a sero-purulent character. Thus while the rheumatic form of pericarditis is, comparatively speaking, a mild affection, and we seldom have to record a fatal termination, where no complication exists, the idiopathic form and that which is consecutive to other diseases, are always formidable and not unfrequently fatal affections.

*The pulse* seldom presents any remarkable peculiarity in the sthenic form of pericarditis; as in other inflammatory affections, it is, as a general rule, accelerated, sometimes considerably. In several of Dr. Graves'\* cases, however, "the pulse was not quicker than natural, from the beginning to the end of the disease; "a perfectly natural pulse (he observes) is not an unfrequent occurrence in pericarditis." He has also twice met with "a sudden decrease in the frequency of the pulse at the very origin of the disease." In this form of pericarditis, the pulse is likewise almost always regular. Dr. Graves, however, says that in many cases of acute rheumatism he has observed irregular action of the heart to be the first signal of the approaching pericarditis; "a weak, irregular, and intermitting pulse may exist (he says) in the very commencement of pericarditis, showing that this kind of pulse may precede effusion, and that it does not necessarily arise from the impediment which the latter must throw in the way of the heart's action."

In the asthenic form of pericarditis, if the movements of the organ are impeded by liquid exudation, the radial pulse as a general rule become small, weak, or intermittent; if the amount of fluid is very considerable, it becomes indistinct or imperceptible. It presented these characters in six out of eight cases given by Corvisart, in five out of eight cases given by Andral, and in three out of four given by Louis; indeed the latter writer looked upon irregularity of the pulse as one of the commonest symptoms of pericarditis. Dr. Graves and others have likewise called attention to "a disparity between the energy of the heart's action and the strength of the radial pulse" as a symptom in pericarditis, the "pulsations in the cardiac region being violent, while the pulse is weak and thready at the wrist."

*Expression of Countenance.*—The countenance in acute peri—

\* Clinical Medicine, vol. ii.

carditis is very generally expressive of anxiety and distress, indeed it sometimes acquires this character very early ; and, when pericarditis arises during the course of acute rheumatism, it is frequently so characteristic as at once to attract attention, and to lead to an examination of the central organs of the circulation, although the patient may complain of no symptom referrible to it. This has been explained by “the sympathy which exists between the respiratory nerves of the face, and those of the heart;” “the impression being conveyed to the spinal cord through the pneumogastric nerves, and reflected to the face through the portio dura.” If we except this expression of anxiety and distress, there is nothing pathognomonic in the countenance in pericarditis; sometimes the face is pale and haggard, sometimes it is heavy and sunken; at others, the features are sharp and contracted, the lips are pallid, the colour is sallow, and the face is bedewed with perspiration; though to this there are many exceptions. The spirits are generally depressed, there is more or less prostration, and the patient is fretful or desponding. In complicated cases, particularly where the disease is associated with inflammation of the lungs or pleura, or where a large amount of fluid is effused into the pericardial sac, the face becomes congested, sometimes œdematous, the lips and cheeks have a livid or purple hue, the patient suffers great distress from dyspnœa, which is depicted in his countenance, and he succumbs under symptoms closely resembling those which accompany the advanced stages of some forms of valvular disease, preserving his intellect generally perfectly to the last.

*Posture of the patient.*—The position which a patient, labouring under acute pericarditis assumes in bed, varies according to the nature of the inflammatory exudation, its amount, and the presence or absence of dyspnœa, though this is by no means a general rule. In the most frequent form of the disease, where the pericarditis arises during the course of acute rheumatism, and where the inflammatory exudation consists almost wholly of lymph, the patient can lie in any position; he almost always, however, prefers that upon the back, which is likewise, we know, the usual position in acute rheumatism; but, he is generally more restless and uneasy than when the rheumatic fever is uncomplicated. When the inflammatory exudation consists principally of liquid, restlessness and uneasiness are prominent symptoms; the patient

sometimes is able to rest upon the back, sometimes upon the left or right side; while often he cannot bear the recumbent posture but is obliged to sit up and to lean forward, with the hand pressed perhaps against the affected side; and if he fall asleep, he is quickly awakened by a sense of impending suffocation. Mr Mackenzie,\* who had the opportunity of seeing many cases of the hæmorrhagic form of pericarditis in Russia, states that when the effusion was limited to the pericardium, the patients preferred to have the head low; some preferred lying upon the face, inclined to the left side, so as to make the region of the heart the most depending part, and to take the weight off the diaphragm; while others lay upon the back. After death, in some of those cases, I found from six to ten pounds of fluid in the pericardium, which occupied nearly the whole of the chest. He mentions one case where the patient could lie equally well in all postures, yet after death the pericardium occupied the whole of the left side of the chest, from the clavicle to the diaphragm, and contained ten pounds of thick brown fluid.

Although restlessness and uneasiness are often present, it sometimes happens that the patient shows the greatest reluctance to any change of posture; he preserves constantly the same position, appearing to fear to make the slightest movement. “While the immobility with which he retains one position would lead us to suppose that any other would be intolerable to him, yet I have known, Dr. Macleod† observes, such a patient upon being moved remain in his new position apparently as determinedly as he previously done in the other.” When pericarditis is combined with pleuritis, and there is at the same time effusion in the pericardium and pleuræ, the patient for obvious reasons, is obliged to lie down; but when the effusion is limited to the pericardial sac, even though its amount is considerable, the patient is sometimes easiest in the supine posture; he seldom lies upon the left but occasionally upon the right side.

#### LOCAL SYMPTOMS OF PERICARDITIS.

*Palpitation.*—When we consider the intimate anatomical union between the pericardium and the heart, we can understand that when the former becomes the seat of acute

\* Lancet: April, 17, 1841.

† On Rheumatism

mation, it must have some influence upon the motions of the latter ; hence, in pericarditis the action of the heart is generally increased, sometimes considerably, while its motions are quickened ; and although the patient may not be sensible of this himself, and may make no complaint of palpitation, it is generally perceptible on laying the hand on the præcordial region. Indeed, increased action of the heart, suddenly ensuing during the course of acute rheumatism, is often one of the first symptoms which attracts attention to the cardiac complication ; and Dr. Graves† has further shown that “the heart’s action sometimes becomes increased in strength for many hours before any physical sign of pericarditis can be detected, and before any pain is felt in the region of the heart.”

In the *sthenic* form of pericarditis, the action of the heart, although quickened, is usually regular, and the impulse is much stronger than natural. Occasionally, however, we meet with cases where the heart’s action at the very commencement is tumultuous, confused, and violent ; while other cases again occur where the impulse is not in the slightest degree increased, although all the physical signs of pericarditis are well marked. When palpitation is a very prominent symptom, it will often be found that the pericarditis is complicated with inflammation of the lining membrane of the heart. In the *asthenic* form of pericarditis, when liquid exudation occurs, the heart’s impulse, as a general rule, becomes weaker in proportion to the amount of the effusion, and sometimes unequal or irregular ; when the amount is very considerable the impulse is quite imperceptible, and the sounds at the same time become indistinct. The cause of the palpitation in pericarditis lies most probably in the increased irritability and consequent disturbance of function which inflammation of the investing membrane occasions in the organ which it covers ; for the same reason that inflammation of the pleura gives rise to cough, and inflammation of the peritoneum to vomiting.

*Pain.*—Pain is a common and sometimes an urgent symptom in pericarditis. Occasionally, however, it is merely a sensation of weight, uneasiness, or constriction, and cases undoubtedly occur where pain is entirely absent. The pain is usually aggravated by pressure, by a full inspiration, or by coughing, often by pressure

† Clinical Medicine, vol. ii.

in the epigastrium, or by percussion of the præcordial region, that the patient cannot bear this manipulation; sometimes increased by motion, by using the left arm, or by lying on the left side. Occasionally the disease sets in with sudden burning pain, referred to the præcordial region, so severe as to make the patient scream; indeed in very early life, pericarditis is sometimes indicated only by the screaming of the infant. Severe pain, as a general rule, is to be regarded as an unfavorable sign. The character of the pain varies; it is sometimes compared by the patient to a tight cord bound round his chest; and it is a burning or lancinating pain which lasts but a short time and frequently returns; sometimes it is attended by a sense of numbness in the left arm, or the pain shoots towards the shoulder and arm. In a remarkable case related by Dr. C. where a hepatic abscess opened into the pericardium, giving rise to pericarditis, the patient was suddenly seized with "acute pain in the cardiac region, followed by violent beating of the heart and burning heat below the left breast." Dr. Latham† says that if it occur at all, almost always occurs early: he has seen the pain when the disease was of little severity, of short duration, and of easy cure; and he has known the severest attack of pericarditis to pass through all its stages without pain.

The præcordial region is the most usual situation of the pain, next the epigastrium, or it extends from one to the other, to the left scapula, or shoulder or arm on that side, seldom ever, reaching below the elbow. It is generally increased by pressure upon the intercostal spaces, or at the epigastrium. Abercrombie‡ first called attention to the latter as a sign of pericarditis. After detailing two cases in which it was present, he observes: "the violent pain produced by pressure on the upper part of the abdomen in these cases is a symptom deserving particular attention: it seems to have been occasioned by the diaphragm pressed upwards against the inflamed parts." Dr. Elliot places particular stress upon "the extension of the pain from the epigastrium to the heart to the scapula, shoulder, and a certain way down the arm (a symptom which patients will not always mention when questioned respecting it), and its increase on strong pressure

\* Clinical Medicine.

† Lect. on Diseases of the Heart.

‡ Trans. of Med. Chir. Soc. of Edinb., vol. i., 1824.

or between the ribs and cartilages over the heart, and upwards under the cartilages of the left false ribs." Dr. Mayne\* looks upon epigastric pain or tenderness as the most unequivocal general symptom of pericarditis; out of eleven cases of the disease, it was present in ten, and in five it was the most prominent symptom. "The tenderness was confined principally to the left side of the epigastrium, and was most intense when the pressure was directed up towards the pericardium underneath the false ribs." Louis says that præcordial pain is met with in about half the cases of pericarditis: out of eight cases given by Andral, pain was present in three, but in those it was very severe.

Although instances do occur where pain is altogether absent, these are to be looked upon as exceptions to the general rule; I have seen very few cases of acute pericarditis unattended at some period by pain, whether the disease was partial or general, or whether it was idiopathic or rheumatic; sometimes it was not complained of unless upon pressure or percussion of the præcordial region; indeed, the pressure of the stethoscope during auscultation sometimes causes pain, particularly if the auscultator is inexperienced and makes unnecessary pressure. Dr. Law† mentions a case where effusion into the pericardial sac occurred suddenly during the course of acute rheumatism, without any preceding or accompanying pain; "it was only when the extent of dulness diminished, that the patient complained of the inconvenience of the heart's action; the organ seemed to have more room to act according as the effusion disappeared." Dr. Stokes‡ on three occasions met with pericarditis in subjects in whom the heart was displaced by pleuritic effusion, and the disease was latent in all of them; with the exception of the attrition murmur, there was (he says), "neither pain, palpitation, nor irregularity of the heart's action preceding nor accompanying the attack."

The cases of acute pericarditis in which pain was altogether absent, seem to have been, for the most part, examples of the disease occurring in the course of acute rheumatism, and setting in when the inflammation in the joints was at its height; its apparent absence under such circumstances can be accounted for by the pain in the joints being so severe as to mask the other; the patient,

\* Dub. Jour. of Med., May, 1835.

† Dub. Jour. of Med., vol. xvi.

‡ Dub. Jour. of Med., vol. xvii.



consequently, unless closely questioned, makes no complaint of comparatively lesser pain in the præcordial region. Indeed, we know that previous to the discovery of the intimate relation between acute rheumatism and pericarditis, it was the rule for pericarditis to pass through its stages without the practitioner's attention being at all attracted to the heart. Besides, some subjects are more tolerant of pain than others, some will bear very severe pain without complaining, while others will be loud in their expressions of pain, although it may be comparatively trifling.

*Cause of the pain.*—Writers differ somewhat in their opinion of the cause of the pain. M. Bouillaud refers it to pleuritic complication; undoubtedly, pleuritis does occasionally complicate pericarditis, but cases are sufficiently numerous where the pain was severe, and yet no pleuritis existed. M. Gendrin refers the pain to the friction of the opposed surfaces of the pericardium during the motions of the heart; but pain is still felt, although an amount of fluid sufficient to separate the two layers of the pericardium, has been effused. By others, again, the pain has been referred to the stretching of the pericardium during the motion of respiration; but if this were its sole cause, it ought to be only in inspiration, which is not the case. It appears a more probable supposition that pain, when severe, depends upon the tension of the inflammation from the serous to the fibrous layers of the pericardium; and that as long as the inflammation is limited to the serous portion of this membrane, pain will not be a permanent symptom.

The pain of pericarditis has been mistaken for that of pleuritis. In the latter affection, however, the respiration is shorter and hurried, and the pain is more remarkably increased on inspiration. It seldom, likewise, has its seat in the præcordial region, usually the lateral and posterior regions of the chest. Again, if a pain is experienced in pleuritis, its seat is towards the inguinal or lumbar region of the affected side; whereas in pericarditis remote pain is usually felt in the left shoulder or arm, or in the left hypochondriac or epigastric regions.

*Dyspnœa* is a symptom, to which, previous to the discovery of auscultation and percussion, more importance was attached than at present; and as it is common to many diseases of the lungs and heart, it is not one of much value. In that form of pericarditis



where the inflammatory exudation consists solely of lymph, a sense of weight, oppression, or tightness in the chest, not amounting to dyspnoea, accompanied or not by cough, is often experienced by the patient; and if dyspnoea is a prominent symptom, it usually arises from the pericarditis being complicated with inflammation of some of the other contents of the thorax. I have, however, seen cases where the dyspnoea from the moment of the attack was extreme, amounting to orthopnoea, although apparently the disease was not complicated with inflammation of any other part, and before effusion, to any amount, had occurred. The breathing under such circumstances is hurried, the respirations are frequent, and the dyspnoea has often an irregular, spasmodic, or paroxysmal character. Although the "respiration is sometimes hurried, this is not uniformly the case;" Dr. Wells has given a case where, although the pulsations of the heart were 190 in the minute, the respirations were not above 24.

In uncomplicated pericarditis, dyspnoea is seldom a prominent symptom, unless a large amount of liquid is effused; this may take place quickly, indeed it has been known to occur within twenty-four hours from the invasion of the disease, and the more rapidly it occurs, and the larger its amount, the more urgent in general will be this symptom. The cause of the dyspnoea here is, in a great measure, mechanical, the distended pericardial sac impeding the motions of the heart, preventing the elevation of the diaphragm, and compressing the lung. It is probable, likewise, as suggested by Dr. Sibson,\* "that an injurious pressure is sometimes exerted on the trachea at its bifurcation, and that this circumstance in part accounts for the extreme dyspnoea so frequently present in cases of pericarditis."

*Orthopnoea* is a doubtful sign in pericarditis. Dr. Taylor has reported several cases where liquid effusion existed without orthopnoea, and three where orthopnoea was present although no liquid effusion existed. This symptom has been already alluded to under the head "position of the patient."

If the foregoing symptoms were present in every case of pericarditis, and if they were always met with in the order in which they have been described, little difficulty would be experienced in the diagnosis of the disease. This, however, is very far from being

\* Lond. Jour. of Med.

the case; sometimes only two or three of them are present, sometimes all, with the exception of inflammatory fever, are absent and the diagnosis must be made almost entirely from the physical signs presently to be considered.

In addition to the symptoms already mentioned, some others are occasionally present, particularly in the idiopathic form of pericarditis, or where inflammation of the pericardium is complicated with inflammation of other tissues—viz., cough, vomiting, hiccup, difficulty of deglutition, and syncope. Cough is not a necessary attendant upon acute pericarditis, unless it is complicated with bronchitis, pleuritis, or pneumonia; neither is it of the chronic form of the disease unless congestion of the pulmonary tissue ensues; it is not, however, a very unfrequent symptom: it is usually dry, and has a spasmodic character. Sometimes it is slight, and others it is very distressing, and teases and harasses the patient all through.

If vomiting is a prominent symptom, there is generally more or less pain at the epigastrium. In such cases, the disease (as Dr Copland observes) “might be mistaken for gastritis, and the singultus and restlessness, the rapid, weak, and irregular pulse, the cold sweats, &c., may be attributed to the unfavourable termination of the latter malady.” A disposition to syncope in the advanced stages of pericarditis is not uncommon; but (as Dr Macleod observes) “the patient very rarely does actually faint.” Syncope is mentioned by Louis as having been present in two out of thirty-six cases, and a tendency to syncope in two others.

#### ANOMALOUS SYMPTOMS IN PERICARDITIS.

Scattered through the periodicals, and in the Transactions of Societies, occasional cases are met with, where acute pericarditis presented anomalous symptoms, viz., where none of its ordinary signs existed, and where attention was withdrawn from the heart by urgent symptoms apparently belonging to disease of other organs or tissues. Thus, Portal reports two cases, which during life were regarded as examples of inflammation of the larynx or trachea; on examination after death, however, these parts were found to be healthy, while the pericardium was acutely inflamed. In other cases, and these are the most numerous, the symptoms were apparently those of inflammation of the membranes of the brain, or spinal

rd. Dr. Watson\* records several examples of this kind, but it is to Dr. G. Burrows that the profession is principally indebted for information under this head; in his Lumleian lectures, and afterwards in a distinct treatise,† he has collected the cases given by various writers, amongst others by Drs. Bright, Abercrombie, Stanley, Latham, Bouillaud, Macleod, Rostan, Andral, and Macintosh, to which he has added some met by himself, where pericarditis simulated: 1. Inflammation of the brain and its membrane; 2. Mania and dementia; 3. Apoplexy and epilepsy; 4. Tetanus and trismus; and 5. Aggravated chorea and hysteria. In many of these cases the inflammation was not limited to the cavity of the pericardium, but extended to the exterior of the sac, to the pleura covering the diaphragm, involving the phrenic or pneumo-gastric nerves upon the left side; or to the endocardium, or muscular tissue of the heart; and in several of the cases it arose during the progress of acute rheumatism.

#### CONCLUSIONS FROM THE LOCAL AND GENERAL SIGNS.

To take a review of the symptoms which have been enumerated, pericarditis evidently cannot be diagnosed with certainty from the constitutional and local signs; it may be strongly suspected, but without the assistance of the signs furnished by auscultation and percussion, a positive opinion could not be pronounced. Among the general signs, inflammatory fever is always present, but if pericarditis sets in during the course of rheumatic fever this symptom can have no value. The local and constitutional signs upon which I lay greatest stress are:

1st. Palpitation or any unusual action of the heart arising *suddenly* in a patient who had not been previously the subject of it. If this occurs to a patient labouring under acute articular rheumatism, it is more certainly diagnostic.

2nd. Any alteration in the pulse, such as its becoming suddenly quicker, or slower, or intermittent.

3rd. A more anxious expression of countenance than usual.

4th. Pain or any uneasy sensation referred to the præcordial region; or if percussion or pressure at this part, or in the epigastrium occasions pain.

\* Lectures on the Practice of Physic.

† On Disorders of the Cerebral Circulation.

If, with the foregoing symptoms, any difference in the auscultatory signs is observed; if a friction sound however slight is heard or the præcordial region yields a more dull sound than natural over an abnormal extent of surface, we need have little hesitation in pronouncing the disease to be inflammation of the pericardium.

#### PHYSICAL SIGNS OF PERICARDITIS.

The physical signs of pericarditis, or those furnished by inspection of the chest, by palpation, by percussion, and auscultation, are of far greater assistance in its diagnosis, and of much more value for determining the form or stage of the disease, than the general or local signs; indeed, previous to the discovery of the two latter it was scarcely possible to distinguish pericarditis from several other affections; and without their aid, we could not in any case pronounce a positive opinion.

#### SIGNS FURNISHED BY INSPECTION OF THE CHEST.

By the eye we ascertain whether the two sides of the thorax are symmetrical, and we detect enlargement, prominence, or bulging of one side; by means of it also we determine the point at which the apex of the heart beats, and we can, in some measure, judge of the strength and extent of the impulse, but less certainly than by the application of the hand. Inspection of the chest is of less value as an aid to diagnosis in the early, than in the advanced stage of pericarditis; and, in cases where the exudation consists solely of lymph than where it consists principally of liquid.

From what has been said of the position of the pericardial sac, and of its relation to surrounding parts, it is obvious that when liquid is effused in such amount as to distend considerably its cavity, the lungs from the nature of their tissue will be compressed and pushed aside, more particularly the left lung; the central tendon of the diaphragm will be depressed, and the stomach and left lobe of the liver will be protruded downwards, occasioning a prominence in the epigastric region, and elevating the xyphoid cartilage; "the hollow which naturally exists at the ensiform cartilage is not only effaced, but there is a more or less considerable projection here."\* If we have the opportunity of making an examination, the central tendon of the diaphragm instead of being con-

\* Avenbrugger translated by Corvisart.

above, will be found to be convex below, and more than an inch lower down than natural, “and the liver and stomach will be displaced downwards in exact proportion to the amount of pericardial distension.”\* Dr. Sibson† has given a drawing taken from a case where the pericardial distension was very great, which shows well the extent to which the liver and stomach may be protruded downwards under these circumstances. Mr. Adams‡ has reported a case where the depression of the liver caused by the distended pericardium, occasioned an unusual prominence in the abdomen, giving rise to a suspicion of enlargement of that organ. The patient was a scrofulous girl of fourteen years of age; the pericardial sac contained twenty ounces of fluid. Mr. South§ mentions another where purulent effusion into the pericardial sac was accompanied by swelling at the epigastrium, and on the supposition that it was an abscess of the liver, a puncture was made, and the pus discharged; after death, the disease was found to have been seated in the pericardium.

When liquid exudation is abundant, particularly in young subjects in whom the cartilages of the ribs are not ossified, the lower portion of the sternum, the costal cartilages, and lower ribs on the left side will be protruded outwards, occasioning a bulging or vaulting of the præcordial region, which is visible to the eye, and can be determined by mensuration. M. Louis|| was the first to call attention to this as a sign of pericarditis. It is not of course an early one, and it cannot obviously occur unless the amount of liquid exudation is considerable. In the case in which it was first noticed by M. Louis, the pericardial sac contained a pint and a-half of liquid. M. Valleix,¶ who regards this as an important sign of pericarditis, describes it as of an oval shape, the long diameter from above downwards, bounded above by the second or third rib, below by the eighth or ninth ribs. At the same time, the intercostal spaces become (he observes) widened by the raising of the ribs, and their concavity is effaced, or replaced by a slight convexity. “The thoracic projection is greatest, according to Dr. Sibson, from the fourth to the seventh left costal cartilages, these parts being over the centre of the pericardium. The prominence,

\* London Jour. of Med., vol i.

† *Ibid.*, vol. i.

‡ Dub. Hosp. Rep., vol. iv.

§ Chelius' Surgery, trans. by South.

|| Memoires Anatomico-Pathologiques.

¶ Guide du Médecin Praticien, tome i.

however, extends in proportion to the distension, over half or two thirds of the sternum—over the left costal from the second downwards, those cartilages being a further apart, and the intercostal spaces widened—over cartilages to the right of the lower end of the sternum left ribs, in the neighbourhood of and outside the nipple fifth or sixth to the seventh or eighth, and over the xiphoid cartilage, the epigastrium, and the seventh and eighth left ribs. M. Gendrin\* does not consider bulging of the region, or tumefaction in the epigastrium, to be due to the presence of liquid in the pericardial sac, but to paralysis of the intercostal and intercostal muscles; an effect of the adjacent inflammation. He has observed it previous to the occurrence of effusion in that form of the disease in which the exudation is composed of lymph alone.

A prominence of the præcordial region is not, however, necessarily a sign of pericardial effusion; it may arise altogether independent of it, particularly in young subjects who have long been under cardiac disease. In the cases of the kind which I have seen, the deformity had been slowly and gradually developed. In hypertrophy with dilatation of the ventricles, the result of chronic disease, commencing early in life, which could be traced to an injury of the chest, or to an attack of acute rheumatism complicated with cardiac symptoms. Another effect of the distension of the pericardial sac (to which attention has been called by Sibson†) is a protrusion of the apex of the heart upward, so that the seat of the impulse is elevated, which is then felt at a higher intercostal space, sometimes even higher up. When the effusion becomes so considerable as completely to separate the parietes of the chest, all visible impulse ceases as a matter of course.

In some cases of pericardial effusion, an undulatory movement is perceived in the intercostal spaces of the upper part of the side of the thorax. This was first noticed by Senac, and is due to the presence of liquid agitated by the movements of the chest during palpitation. Dr. Latham‡ says it is limited to the intercostal spaces between the second and third or the third

\* Leçons sur les Mal. du Cœur.

‡ Clin. Lect. on the Heart.

† Trans. of Prov. Association.

ribs, and that a vibratory motion, perceptible to the hand, is always felt at the same points. Dr. Sibson\* is of opinion that this phenomenon is not due, as Senac supposed, to the agitation of fluid in the pericardial sac, but has its cause in the slight and peculiar impulse of the heart. This undulatory movement cannot by any means be regarded as a diagnostic sign of the presence of liquid in the pericardial sac. I have seen it in cases of cardiac disease, where undoubtedly no effusion existed. Dr. Taylor† also has observed it, not only when fluid was present, but in enlargement of the heart without effusion, and in adhesion of the pericardium.

#### SIGNS FURNISHED BY PALPATION.

In acute pericarditis of the sthenic form, the impulse of the heart is very generally increased, so much so as to be sometimes distressing to the patient; indeed, in acute rheumatism, this not unfrequently is the first symptom which draws the patient's or practitioner's attention to the heart; cases, however, occasionally occur where we cannot detect any increase of impulse, although the other signs of pericarditis are well marked. When the impulse is much increased, or the heart's action is irregular at the outset, the pericarditis is very generally associated with endocarditis.

Accompanying increased impulse, a vibratory tremor or a sensation of friction perceptible to the hand laid on the præcordial region, is not unfrequent, which resembles, in a slight degree, the fremitus of certain forms of valvular disease, but is not so strong, has a totally different source, and presents much more of a friction character. This, the tactile vibration or "pericardial friction fremitus" was first described by Dr. Stokes,‡ and was shown by him to be due to the presence of lymph upon the opposed surfaces of the pericardium. Whenever it is felt, a friction sound will be audible on auscultation over the same part of the præcordial region, and the presence of a moderate amount of liquid effusion does not necessarily cause its total disappearance. "It is of shorter duration than the accompanying friction sound; a more powerful rubbing is required to render friction of membranes a tactile than an audible phenomenon." When the friction sound ceases, this sign is no longer observed.

\* Lond. Jour. of Med., vol. i.

† Dub. Jour., Sep., 1833.

‡ The Lancet, 1845-46.



When the inflammatory exudation consists principally of liquid and the heart is otherwise unaltered, the impulse will be felt upon a higher level than natural, because as the liquid gravitates to the most dependent part of the sac, the heart is suspended in, and floated upwards in some measure by it. M. Skoda\* lays it down that as the heart is specifically heavier than the effusion, it will gravitate towards the lowest part of the pericardium, so far, at least, as its attachments permit, the fluid occupying the upper part. When the patient is examined in the recumbent posture this undoubtedly occurs, and we are thus enabled by percussion to detect the presence of fluid in the pericardial sac; but when the patient is examined in the sitting posture, the liquid will gravitate to the lower part of the sac, and the heart being supported above will be floated upwards by it, and its impulse will consequently be felt upon a plane higher than natural. When the amount of liquid exudation is still more considerable, the impulse becomes weaker, undulatory, or unequal; and if the amount is so large as completely to separate the heart from the parietes of the chest, all impulse necessarily ceases.

Manual examination of the præcordial region, we thus see, is capable of affording considerable aid in the diagnosis of pericarditis, and should be employed daily in every case of acute articular rheumatism. "If it were the ordinary practice (as Dr. Elliotson remarks, and Dr. Hope repeats,) to place the hand on the præcordial region in all severe inflammatory affections, in the same way that we daily feel the abdomen in fever (even though the patient makes no complaint), we should seldom fail in detecting increased impulse, or some other anomaly in the heart's action; for there can be little doubt that in many cases in which its symptoms were supposed to be absent, they were not so, but were masked by others of predominant severity."

#### SIGNS FURNISHED BY PERCUSSION.

In the sthenic form of pericarditis, and where the inflammatory exudation consists altogether of lymph, the region of the heart's superficial dulness can scarcely be said to be altered. Indirectly, however, percussion of the præcordial region often becomes a useful diagnostic sign, owing to the pain this manipulation

\* Treatise on Auscultation and Percussion.



occasions, which, in some instances, is so severe that the patient will not allow it to be repeated. In the asthenic form of pericarditis, where the inflammatory exudation consists chiefly of liquid, the limits of the heart's superficial dulness on percussion are always increased, sometimes considerably; and in proportion to the amount of the effusion, so will the region which yields a dull sound be extensive, or the contrary.

It has not been exactly determined what is the smallest quantity of liquid effusion into the pericardial sac which can be detected by percussion. Dr. Walshe says "four ounces will widen the area of dulness," which we much doubt. M. Louis considers from eight to ten ounces to be sufficient to produce this effect; in the majority of the cases given by him, however, it exceeded this. In a case recently under my care, where pericarditis supervened upon pneumonic abscess of the left lung, and the pericardial sac contained about eight ounces of liquid, no increased extent or amount of dulness, on percussion, was perceived in the præcordial region on a careful examination of the part the day before the patient's death, which was sudden. When the amount of liquid exudation is trifling, it may gravitate to the most dependent part of the sac, and thus escape detection; when, on the other hand, it is sufficient to distend the pericardial sac, the lungs will be pushed aside, and a large space will yield a dull sound on percussion. A source of error might, however, arise here if old adhesions united the pleura on each side in front of the pericardium. The dulness is at its maximum, according to M. Valleix, on a level with the fourth ribs, where a marked resistance, M. Piorry observes, is also perceived.

The shape of the space which yields the dull sound is peculiar; the pericardial sac, we have seen, has a pyriform shape, the base below, the apex above, exactly the reverse of that of the heart *in situ*; hence, when it is distended by fluid, the region which yields a dull sound will be wide below and diminish gradually in width as we ascend towards the neck. We have likewise seen that the pericardial sac naturally extends to a level with the articulation of the second left rib with the sternum; indeed, I have found it in the healthy subject to reach the level of the first rib. Hence, the site of the dulness will rise much higher in cases of extensive pericardial effusion than in any morbid condition of the heart; and it

may reach the upper edge of the sternum and the left clavicle, and rise above the latter. Dr. Sibson\* has found, by artificially distending the pericardial sac, that when the amount of liquid is not very considerable, the highest point of the sac presents a peculiar peaked shape; and that when the amount is very large, the pyriform figure of this space is lost, it acquires rather a globular shape, the transverse exceeding the vertical diameter.

As the effused fluid is absorbed, whether under treatment or otherwise, the space which yielded a dull sound on percussion diminishes from above downwards; the sounds of the heart which have been feeble, remote, or muffled, according to the amount of effusion, become again audible and distinct, and the attrition sound which had been obscured by the presence of fluid, or prevented from taking place if it was large, are again heard.

Pericarditis is not the only morbid state in which the region of the heart's superficial dulness is abnormally extensive; when the heart itself is considerably enlarged, a larger surface than natural in this region will yield a dull sound on percussion. The shape of the space over which the dull sound is elicited is not the same, however, in the two affections; while the degree of dulness and the sensation of resistance are likewise different. We have seen that in pericardial effusion the dull space is broad below and narrow above, and rises high towards the clavicle; in enlargement of the heart, on the other hand, the dull space is always on a lower plane, never reaching above the third rib and extends more to the left side; the degree of dulness is likewise more marked, and the sensation of resistance greater on percussion over liquid than over an enlarged heart. These characteristic signs, taken in conjunction with the history of the case, with the rapidity with which effusion takes place, and with the changes which occur in the amount of the fluid from day to day, as indicated by percussion, will always enable us to distinguish between them. Pleuritic effusion on the left side has sometimes been mistaken for pericardial effusion. Such a mistake was not uncommon before the discovery of the physical signs of the two affections; it might even now cause a difficulty in the diagnosis, if fluid were simultaneously effused into the pericardium and left pleura. Indeed, Dr. Addison† thinks that "enormous accumulations of fluid in the pericardium, cannot, by physical

\* Lond. Jour. of Med., vol. i.

† Guy's Hosp. Rep. vol. iv.

signs, be always distinguished from effusion into the cavity of the pleura." Tubercular infiltration of the left lung could scarcely be mistaken for pericardial effusion, though its presence might increase the difficulty of diagnosis.

#### SIGNS FURNISHED BY AUSCULTATION.

The signs furnished by auscultation are more precise and positive than those yielded by any of the other physical methods of diagnosis; and although but a few years comparatively have elapsed since their discovery, their prominent features are familiar to every one who has made auscultation a study. Indeed, at the present day, it seems strange that sounds which are so remarkably characteristic, and at the same time so easily recognized, should have been so long overlooked; or that such experienced auscultators and accurate observers as Laennec, Louis, and Andral, should have failed to detect them.

We are indebted to Dr. Stokes\* for the first complete description of these auscultatory signs. M. Collin† had, it is true, under the name "bruit analogue au craquement du cuir neuf," described a peculiar sound heard by him in pericarditis; and M. Broussais‡ had recognized a sound analogous to that of two dry bodies (as parchment), rubbing against one another; but their remarks were overlooked, or failed to attract attention until the publication of Dr. Stokes's memoir. In the following year, M. Bouillaud's§ description of the attrition sounds of pericarditis appeared; Dr. Watson|| did not publish upon the subject until a year subsequently, when he communicated two cases of pericarditis to the *Medical Gazette*, in which he described the friction sound under the name of the "to-and-fro rubbing sound." Dr. Mayne's¶ paper on the subject closely followed that of Dr. Watson.

We have seen that in the normal state, the opposed surfaces of the pericardium glide over one another, during the motions of the heart, without producing sound; at least any which can be detected by the ear applied to the parietes of the chest. We have

\* Dub. Jour. of Med., Sept., 1833.

† Les diverses Méthodes d'Exploration de la Poitrine, 1824.

‡ Commentaires des Propositions de Pathologie, t. i., 1829.

§ Art. "Pericardite," Dict. de Med. et de Chir., 1834.

|| Lond. Med. Gaz., April, 1835.

¶ Dub. Jour. of Med., May, 1835.

likewise seen that exudation of lymph is a very early result of inflammation of this membrane: the effect of which is to render the opposed surfaces of the membrane uneven or rough, and to occasion a greater amount of friction or attrition between them during the movements of the heart. This increased friction is now known to be capable of developing sound, which varies in intensity in different cases, from the slightest possible shade of rubbing, to a harsh grating sound, and which is sometimes sufficiently strong to communicate a vibratory tremor to the hand laid upon the præcordial region.

These abnormal sounds have received the names, attrition or friction sounds, or murmurs, from their character; and pericardial, exocardial or peripheral murmurs, from their seat. (The term *murmur*, however, ought, in my opinion, to be restricted to the abnormal sounds developed in the interior of the heart, in the large arteries and veins). These sounds are almost limited to cases of inflammation of the pericardium, where adhesive lymph constitutes a portion of the exudation. They may mask or obscure, but they never interfere with the intrinsic sounds of the heart.

#### VARIETIES OF THE FRICTION SOUNDS.

The friction sounds of pericarditis present various shades of intensity, and the following have received distinct names, principally from this circumstance:—

1st. *The slight friction sound*—"bruit de frôlement" of the French—is a soft, grazing, or rustling sound, audible only at the commencement of each systole or diastole of the ventricles; it resembles the grazing sound produced by gently rubbing together the palms of the hands, or when louder, the rustling sound caused by crumpling the paper of which bank notes are made, or the rustling of silk.

This sound is supposed by some to have its cause in inflammatory injection of the serous membrane; but Dr. Williams' experiments prove this to be incapable of developing a friction sound. Others refer it to a dry state of the opposed surfaces of the pericardium; the earliest effect of inflammation being supposed to be the suspension of the natural exhalation, which, in the normal state, lubricates the serous surfaces. But do these surfaces

\* Proceedings of Brit. Association.

ever become dry? If, as an effect of inflammation, exhalation is checked, absorption at the same time will be suspended, and the cavity of the pericardium being a shut sac, evaporation cannot take place. I am of opinion that this slight friction sound has its cause in the presence of a very thin film of lymph upon the visceral layer of the pericardium alone, or if upon both surfaces, the deposit occupies a limited portion only, or the back of the sac. Indeed the interval which elapses between the invasion of the disease and the exudation of lymph, is extremely short; and as Dr. Macleod\* remarks, "there is no evidence of the degree of dryness of the membrane ever occurring, which is assumed to take place as the result of inflammatory action and to give rise to a friction murmur; while we have proof positive of the surfaces becoming rough."

2nd. "*The rasping sound*"—the "bruit de frottement," "bruit de râpement" of French writers, the "to-and-fro" sound of Dr. Watson, has, as its name denotes, a rasping, grating, or rubbing character; it is more common than the preceding, which usually soon passes into it. This sound sometimes accurately resembles the rasping or sawing of wood, or the grating of a nutmeg, and it may be imitated in a variety of ways. The sound which it struck me as most nearly resembling, in some cases, was that which would be produced by scratching with the nail the surface of a dry bone. There is no difference of opinion as to the mode of production of this sound, which is always due to the presence of false membrane; and according to the amount of lymph, and its qualities, and as the action of the heart is vigorous or the contrary, it will vary in roughness and intensity.

3rd. *The sound resembling the creaking of new leather*—the "bruit de cuir neuf" of M. Collin, resembles pretty accurately the creaking of a saddle, but is not so loud. Dr. Copland, however, mentions a case where it was heard without the assistance of the stethoscope, and was audible also to the patient, who compared it to the creaking of new shoes. This sound was supposed by M. Collin to have its cause in a dry state of the opposed serous surfaces; but it is now known to depend upon the presence of false membrane; usually in the form of a thick layer of adhesive lymph which has formed partial adhesions, and which are stretched

\* Treatise on Rheumatism.

during the alternate movements of the heart. According to Dr. Copland,\* it may have its cause in a thickened or condensed state of the serous and subserous tissue of the pericardium, especially of that portion reflected over the heart, and the formation of a dense and elastic false membrane. In a case given by Dr. Taylor,† where this sound was heard, both surfaces of the pericardium were everywhere coated with soft lymph. In another, where the friction changed to a new-leather creak, signs of slight liquid effusion occurred at the same time.

4th. *The continuous rumbling murmur* of Dr. Hope, has a hollow, rumbling character, and is audible only in one particular morbid state—viz., when a small amount of liquid is contained in the pericardial sac, the surfaces of which are at the same time coated with lymph; and the sound is produced by the agitation or churning of the liquid during the motions of the heart. In a recent case, where a portion only of one surface of the membrane was coated with lymph and a small amount of liquid was contained in the sac, the heart being at the same time enlarged, the sound, though nearly continuous, had not a rumbling character, but was a moist friction, or crepitating sound, resembling that heard in certain diseased states of the lungs.

5th. *The clicking murmur* of Dr. Walshe.‡ “Occasionally,” Dr. Walshe observes, “sounds are heard of a peculiar clicking character (only one or two with each beat of the heart) which are only distinguishable, at the time, from modifications of the valvular sounds by their non-synchronism with these, and by the extreme irregularity of their occurrence.” “I have satisfactorily traced these clicks (he says) to the pericardium, and further, in all probability, to the separation (without attrition) of surfaces glued together with exudation matter.”

6th. *Murmur produced by bending of layers of exudation matter.* “It has appeared to me (Dr. Walshe observes), that sound is sometimes generated in layers of false membrane, though so perfectly agglutinated together that attrition or separation of the opposed surfaces is physically impossible.”

In the only positive instance of the kind observed by Dr. Walshe, the sound was faintly creaking. “The bending and

\* Dict. of Med.

† The Lancet, 1845-46.

‡ Treat. on Diseases of Lungs and Heart.

crumpling of tough false membrane may conceivably, he thinks, generate such sound."

CIRCUMSTANCES WHICH MODIFY THE AUSCULTATORY SIGNS OF PERICARDITIS.

The abnormal sounds now described are almost limited to inflammation of the pericardium, and are pathognomonic of the forms of the disease in which either lymph alone is effused, or in which the exudation consists partly of lymph and partly of liquid. When such an amount of liquid exudation occurs as to keep the opposed surfaces of the pericardial sac *altogether* apart, friction obviously cannot take place, and these sounds necessarily cease. It has not, however, been determined what is the smallest amount of liquid exudation which will prevent an attrition sound from taking place. Dr. Taylor\* mentions a case where it was audible on the day of the patient's death, and on examination the pericardial sac contained eight ounces of liquid. I have heard it within three days of the patient's death, when eight ounces of liquid was found in the sac; in another case, however, it was not audible the day preceding the patient's death, when the same amount of liquid was contained in the pericardium, although the precordial region was carefully examined, and the heart's action was not by any means feeble. Dr. Walshe thinks that a less amount will cause its disappearance; but in another place, he mentions a case where sixty ounces of liquid were found, yet twenty-nine hours before death an attrition sound had been distinctly audible. Other circumstances, however, independent of the exact amount of liquid exudation, must be taken into account, particularly the strength or feebleness of the action of the heart at the time, and the degree or amount of compression which the parietes of the heart suffer from the effusion.

When the pericardial sac contains such an amount of liquid exudation as to cause the disappearance of an attrition sound in the recumbent posture, it may sometimes be brought out by placing the patient in the sitting posture, when the liquid will gravitate to the lowest part of the sac, and the heart being floated upwards by it, the attrition sound may be heard about the base of the organ. Dr. Sibson† has shown that when these sounds become

\* The Lancet, 1845-46.

† Lond. Jour. of Med. vol. i.



inaudible, owing to the presence of liquid exudation, they sometimes be reproduced by making pressure upon the *c* parietes, and so displacing the layer of liquid which intervenes. The extent of surface over which they are audible, the intensity of the sounds, and the variety of shades they present, depend not upon the quantity and quality of the lymph effused, and the extent of surface covered by it, than upon the strength or feebleness of the action of the heart; if its action is very feeble, no abnormal sound may be audible. The presence of lymph upon only one surface of the pericardial sac is sufficient to give rise to a friction sound; I have heard it when the visceral layer of the pericardium only was the seat of the deposit, and when the latter did not exceed the size of a shilling.

As a general rule, these sounds, when fully developed, are double, and accompany both the systolic and diastolic movements of the ventricles; they are also usually louder during the former. Sometimes, however, the friction sound is limited to the period of the ventricular systole, less frequently to that of the diastole, though Dr. Taylor\* says, when single it usually accompanies the diastole. Dr. H. Kennedy† has called attention to the fact, that the pericardial friction sound, on its very first appearance, intermits or does not accompany every ventricular systole; he has observed this to occur on the decline of pericarditis. Dr. Kennedy has likewise shown that a friction sound is often single at first, “before, in fact, the double one is formed; and again, when the double *friction* sound is on the decline, it is not at all uncommon to hear at irregular intervals a single sound.”

The friction sounds of pericarditis usually give the impression of being more superficial and nearer the ear than valvular murmurs. This will be very marked if friction occurs outside the pericardial sac, owing to a deposit of lymph exterior to the pericardium. The sounds will appear more remote and distant when the deposit of lymph is limited to the back of the heart. I have occasionally heard them with the ear at a little distance from the stethoscope, but they seldom, however, entirely mask the ordinary sounds of the heart. In most instances they are audible only in the præcordial region and its immediate vicinity; they may have the same intensity over every part of this region, though they are usually louder

\* Med. Chir. Trans. vol. xxviii.

† Dub. Med. Press, Jan., 1853



at the middle of the sternum, or to the sternal side of the nipple; in some few instances they were audible posteriorly as well as anteriorly. Dr. Graves\* has shown that these sounds will be loud and widely diffused "when pericarditis supervenes upon an enlarged and hypertrophied heart; the rubbing surfaces being then more extensive, and a larger portion of the body of the organ coming in contact with the parietes of the chest." On the other hand, when the heart's action is extremely feeble, no friction sound, or scarcely an appreciable one, may be audible, although the opposed surfaces of the pericardium are coated with abundance of lymph.

Attrition sounds are scarcely ever so prolonged as valvular murmurs, and they seldom quite obscure the normal sounds of the heart; Dr. Graves, however, mentions a case where the only sounds audible were a *bruit de scie* and a musical sound; on the next day the musical murmur was replaced by a loud leather creak. On examination, the surfaces of the pericardium were coated with recent lymph; at the apex the opposed membranes were firmly united; the heart itself was hypertrophied, and both ventricles dilated. In some instances attrition sounds are audible only in the erect or sitting posture, and disappear when the patient is recumbent; in others, they are only heard in the recumbent posture; these, however, are exceptional cases, and it is probable that the cause lies in the deposit of lymph being partial and limited to either the anterior or posterior surface of the sac, or in the presence of a certain amount of liquid exudation in addition. In a case which was in hospital some time since, the friction sound, which was best marked over the upper portion of the sternum, diminished very considerably when the patient lay down, and reappeared in the sitting posture. In this case, on the patient's admission, a considerable amount of liquid exudation existed, and as it was absorbed, the friction sound became developed.

Dr. Sibson has shown that in the earliest stage of pericarditis, before in fact a friction sound is to be heard, it may sometimes be developed by making pressure with the stethoscope upon the præcordial region, so as to bring the two surfaces closer together. "This effect of pressure is rarely excitable (he observes) in men after the age of 45, the cartilages of the ribs being then firm and

\* Clinical Medicine.

unyielding." Dr. H. Kennedy, without being aware of Dr. Sibson's researches, arrived at the same conclusions. The only objection to this proceeding is that, in not a few cases of pericarditis, pain is caused by even the moderate amount of pressure of the stethoscope in auscultation of this region.

Attrition sounds vary in intensity at the same part of the chest at short intervals; they are also usually of short duration; they diminish or disappear under treatment, and subside altogether when adhesion of the opposed layers of the pericardium ensues. They will likewise disappear when such an amount of liquid exudation occurs as completely to separate the two surfaces of the membrane; and as this is absorbed, they again become audible and continue to be so until adhesion ensues. Again it may happen (as Dr. Mayne suggests), "That the lymph which previously coated the membrane breaks down and becomes diffused through the fluid, instead of adhering to the surface by which it was secreted, so that even though the heart and pericardium remain in apposition, no *frottement* could result."

The period which intervenes between the invasion of pericarditis and the development of a friction sound, varies somewhat according to the intensity of the inflammation and the acuteness of the attack. In very acute cases, lymph sufficient to cause an attrition sound has been effused within twenty-four hours; in the majority of cases, between twenty-four and forty-eight hours elapse before it becomes evident. In two of the cases recorded by Dr. Mayne, an interval of three days elapsed before a friction sound was heard.

The duration of the friction sounds necessarily varies, being influenced, on the one hand by the rapidity of the changes which the lymph undergoes, and on the other by the amount and nature of the liquid exudation. According to Zehetmayer, quoted by Dr. H. Davies,\* "the exudation may exhibit symptoms of an organizing process within forty-eight hours from the time of its deposition, and present distinct traces of the presence of areolar tissue within a period of fourteen days." I have found the opposed layers of the pericardium perfectly adherent throughout when the illness was of only twelve days duration, and no friction murmur had been audible from the eighth day. When pericarditis occurs

\* Lect. on the Lungs and Heart, 1851.

in infancy, a shorter period seems to be sufficient for adhesion to take place; in a case of idiopathic pericarditis in an infant seven months old, recorded by Dr. Churchill,\* in which death occurred on the third day, the opposed layers of the pericardium were found to be adherent. Hence, we can understand that considerable difficulty in the diagnosis might arise if the patient was not seen until the inflammation had persisted for a certain number of days, and adhesion had already taken place; auscultation, then, could obviously afford us no aid, and the disease might escape detection. There can be little doubt that this circumstance contributed materially to keep up the obscurity in which the auscultatory signs of pericarditis were so long involved.

A person who has once been the subject of pericarditis, may suffer a subsequent attack; this is *less* liable to occur if the first had been followed by universal adhesion of the pericardium. If, however, the adhesions were partial, the interspaces might become the seat of plastic deposit. It is obvious that, under such circumstances, auscultation could not be of the same assistance to the diagnosis; because if the adhesions were universal, no attrition sound could occur; while if they were partial, any sound developed must be either very feeble, or have little of the attrition character.

Dr. Addison† is of opinion that “auscultation will not always enable us to distinguish a friction sound produced *within*, from a friction sound produced *without* the pericardial sac;” in other words, “friction between the serous surfaces of the pericardial sac, from friction between the loose pericardium and lung, or parietes of the chest.” This, however, must be a rare source of error; and even should the difficulty arise, it can make no difference in the treatment.

#### SOUNDS OF THE HEART IN PERICARDITIS.

The intrinsic sounds of the heart are not, as a general rule, altered in that form of pericarditis in which the inflammatory exudation consists solely of lymph. On the other hand, when the pericardial sac contains much liquid, these sounds become more feeble, distant and deficient in tone, in proportion to the amount of effusion; and when it is very copious, they are quite inaudible. Dr. Taylor gives a case where the sounds of the heart

\* Treat. on the Diseases of Children.

† Guy's Hosp. Rep., vol. iv.

had the foregoing characters, and yet scarcely any liquid effusion existed. He is of opinion that alteration of the first sound may be the result of some modification in the contraction of the muscular fibres of the left ventricle consequent on the adjacent inflammation.

#### BRUIT DE SOUFFLET.

In acute pericarditis, particularly of the rheumatic form, a bruit de soufflet, which replaces the first sound of the heart, more rarely the second sound, seldom both sounds, is by no means uncommon. This is almost always the result, either of old valvular disease, or of recent endocarditis complicating the pericarditis. A bruit de soufflet, however, is occasionally heard independent of any morbid condition of the valves; it accompanies the ventricular systole, and is usually best marked in the large vessels which come off from the arch of the aorta, particularly the carotids. It is observed in anæmic individuals who become the subject of pericarditis; or it may happen that pericarditis is set up in a case of relapse of acute rheumatism, where the treatment of the primary attack had been antiphlogistic, by which the patient had been reduced to an anæmic state, when the same murmur would be audible, leading sometimes to a suspicion that endocarditis complicated the pericarditis.

Independent of anæmia, however, there is one particular condition of the parts, in which a systolic bruit de soufflet may be directly the result of pericarditis. For instance, when the exudation consists principally of liquid, and this is in such amount as to distend considerably the pericardial sac, the aorta or pulmonary artery may be compressed near their origin, their calibre slightly diminished, and a bruit developed, precisely in the same way as where direct pressure is made upon a large artery. "The effusion (Zehetmayer\* observes) contracts the impaired elastic tube of the aorta, and establishes an impediment to the current of the blood at the commencement of that vessel, where its walls are thinned, for the reception of the semilunar valves, and the circular and elastic fibres are deficient." The murmur here is usually weak, and has been observed only when a large amount of liquid, as from one or one and a half pounds, was effused.

\* Davies' Lect. on Lungs and Heart.

CHARACTERS BY WHICH PERICARDIAL FRICTION SOUNDS ARE DISTINGUISHED FROM VALVULAR MURMURS.

The attrition sounds of pericarditis have been, and are still, by inexperienced auscultators, sometimes mistaken for, or confounded with, valvular murmurs. In the great majority of cases, however, the distinctive characters of the two classes of sound are well marked and quite characteristic. We are therefore somewhat surprised at M. Skoda's assertion, that he "knows no sign by which the friction sounds of the pericardium can be distinguished from the internal murmurs of the heart, excepting this, that the internal murmurs correspond pretty exactly to the rhythm and to the natural sounds of the heart; whilst the pericardial friction sounds seem to follow upon the movements of the heart. This distinctive sign is only available when the murmur is somewhat prolonged; if it be of short duration, we cannot determine whether it is endocardial or pericardial."\*

Some difficulty might certainly be experienced in making the diagnosis, if the heart's action was exceedingly feeble; if the pericarditis was complicated with pleuritis, or bronchitis with loud bronchial râles; if the subject was an infant, or dyspnoea was so extreme that a proper examination could not be made. These, however, are exceptional cases; when attrition sounds are present, they may always, in my mind, be distinguished from valvular murmurs by attention to the following rules:—

1. Attrition sounds, as a general rule, give a sensation of friction or rubbing, and are usually rough, grating, or creaking, never blowing. Valvular murmurs, on the other hand, are usually blowing.
2. Attrition sounds are usually double, and the second sound is loudest at the same part of the chest as the first. Valvular murmurs, on the other hand, are usually single; and when double, the point at which each is best marked is different.
3. Attrition sounds are generally loudest over the middle of the sternum, or immediately above the nipple. Valvular murmurs, on the other hand, are often loudest about or below the apex of the heart.
4. Attrition sounds are not audible in the course of the large vessels which come off from the aorta, nor are they heard in general

\* Translation by W. O. Markham, M. D.

much beyond the præcordial region, in both which situational murmurs are frequently audible.

5. Attrition sounds give the impression of being morbid and near than valvular murmurs, and are often accompanied by a fremitus, perceptible to the hand laid on the præcordial region.

6. Attrition sounds are sometimes audible only in the sitting posture, or are developed or increased in intensity when the patient leans forward, or when pressure is made with the stethoscope. Valvular murmurs, on the other hand, usually have the same characters in every position of the patient, and unless in a few exceptional cases hereafter to be noticed, are not increased by pressure upon the præcordial region.

7. Attrition sounds are usually of short duration, and increase in intensity at the same part of the chest at short intervals, and subside under treatment, or subside altogether within a limited time. Valvular murmurs, on the other hand, present the same characters at the same part of the chest for a lengthened period, do not subside readily under treatment, and seldom subside altogether.

8. Attrition sounds may be confused with the normal sounds of the heart, but they do not have the same characters. Valvular murmurs, on the other hand, either replace the normal sounds, or prevent them from being heard.

## CHAPTER XI.

OF PERICARDITIS.—CONNEXION OF WITH ACUTE RHEUMATISM.—  
 ENCY OF PERICARDITIS.—COMPLICATIONS OF.—TERMINATIONS  
 EFFECTS OF.—PERICARDIAL ADHESION.—DIAGNOSIS OF PERICARDIAL  
 ION.—TREATMENT OF PERICARDITIS.—PARACENTESIS OF THE  
 ARDIUM.

ATION of the pericardium may be directly the result of  
 ng wounds or other injuries of the pericardium; or it  
 ow injuries of the chest, as a fall or blow upon the left  
 ericarditis may also arise under the same circumstances as  
 lammation of internal organs generally, such as exposure  
 and moisture when the body is heated, &c. This, the  
 c form of pericarditis, is very generally associated with  
 ition of some of the other contents of the thorax; or the  
 ition may extend to the pericardium from neighbouring  
 r tissues, as the pleura, lungs, &c. It may be also the  
 the irritation excited by cancerous or tubercular deposit.  
 ews\* has recorded three cases of the latter. The presence  
 foreign matters in the pericardium will likewise give rise  
 arditis; Dr. Graves† has related a case where a hepatic  
 ened through the diaphragm into the pericardium, causing  
 lammation of this membrane. A somewhat analogous  
 been recorded since by Dr. Bentley.‡ Dr. McDowel§  
 ntly reported one where a tubercular abcess of the lung  
 nto the pericardial sac, giving rise to pericarditis; several  
 mples of which are also upon record. In cardiac aneurism,  
 tion excited by the growth of the tumour may give rise

arditis occurs occasionally during the progress of, or as a  
 f influenza, or the eruptive fevers, particularly scarlatina

l. Med. Gaz., March, 1847.

l. Med. Gaz., Dec. 1848.

† Clin. Med., vol ii.

§ Pro. of Path. Soc.

and small pox. Dr. Henry Kennedy\* met with it three times scarlatina, and Dr. S. Alison† has also recorded three cases where it supervened upon this disease. It is sometimes observed in connexion with chorea, but generally only when the latter arises in the course of acute rheumatism: the association of these two morbid states was first noticed by Dr. Copland‡; Dr. Bright§ gives five cases where the complication existed; and Dr. Kirke¶ has recently condensed most of the information we possess, under this head.

Pericarditis sometimes arises in the course of erysipelas, more frequently in cases of diffuse inflammation, phlebitis, or pyæmia; it occasionally succeeds severe injuries, or follows capital operations; Dr. Blackiston¶ mentions two cases where it supervened upon amputation of the leg; and Mr. Bransby Cooper\*\* give one where it followed the operation of ligature of the femoral artery from popliteal aneurism. When it occurs under such circumstances, it is probably due to pyæmia. It has followed the simple operation of lithotomy, but here the kidneys were probably unsound. Among the predisposing causes of pericarditis, we may include states of debility succeeding to acute diseases, or fever, and cachectic conditions of the system, however induced; thus we have seen that, in certain localities, a peculiarly fatal form is met with, which is believed to have its origin in scurvy.

The most frequent causes of pericarditis, or, at least, the morbid conditions in which it is most frequently met, are acute rheumatism, and uræmia from diseased kidneys. Dr. Taylor‡ was the first to call attention to the frequent connexion of pericarditis with Bright's disease; indeed, according to him, Bright's disease, in its advanced stage, is almost as frequent a cause of pericarditis as acute rheumatism. This point is also illustrated in the *Decennium pathologicum* of Dr. T. K. Chambers,†† in which he has recorded the results of the examination of the bodies of 216 individuals, who died at St. George's hospital of various diseases—marks of *recent inflammation* were found in the pericardium in 135 subjects.

\* Dub. Med. Press, Jan. 1853.

† Lond. Med. Repository, vol. xv.

‡ Lond. Med. Gaz., 1850.

\*\* Lectures on Surgery, p. 792.

†† Brit. and For. Med. Chir. Rev., vol. xii., 1853.

† Lond. Med. Gaz., Feb., 1845.

§ Med. Chir. Trans., vol., xxii.

¶ On diseases of the Chest.

†† Med. Chir. Trans., vol. xxvii.



The probable direct causes of the inflammation were, according to him, as follows :—

	Cases.
Rheumatic Fever ... ..	18
do with diseased heart and kidneys	1
Uræmia from diseased kidneys (either alone or with diseased heart) ... ..	36
Diseased heart and Dropsy ... ..	18
Pyæmia ... ..	17 or 18
Erysipelas ... ..	4
Petechial or typhus fever ... ..	3
Abscesses after fever ... ..	1
Pneumonia ... ..	10
Peritonitis from accidental causes ... ..	3
Pleuritis or empyema ... ..	5
Vomicæ in the lungs ... ..	8
Malignant Disease in actual contact ... ..	2 or 3
do in the neighbourhood ... ..	1
Neighbouring Abscess ... ..	2
Slough of œsophagus ... ..	1
Aneurism of aorta ... ..	2
Fracture of Sternum, &c. ... ..	1

Although it would appear from the foregoing table, that uræmia is a more frequent cause of pericarditis than acute rheumatism, the fact lies altogether the other way, acute rheumatism being by many degrees the most common cause. We know that the great majority of cases of rheumatic pericarditis recover from the attack, while the majority die when acute pericarditis supervenes on Bright's disease; hence statistical tables of the *deaths* from pericarditis by no means show the frequency of its occurrence in connexion with acute rheumatism as compared with uræmia.

#### CONNEXION OF PERICARDITIS WITH RHEUMATIC FEVER.

The close relation which exists between rheumatic fever and inflammation of the pericardium is comparatively a modern discovery; and for establishing it we are entirely indebted to British physicians. Sir D. Dundas,\* writing in the year 1809, says: "There is a disease of the heart which I apprehend is not very uncommon, and yet I do not believe any account of it is to be found in any medical author." . . . "In all the cases which I have seen, this disease succeeded one or more attacks of *rheumatic*

\*Trans. of Med. Chir. Soc., vol. i., 1809.

*fever.* . . . . All those I have seen afflicted with this disease were young persons, only two being above twenty-two years of age. Seven out of the nine died; of these, six were examined. In all, the heart was uniformly found to be enlarged; in one case, fluid was found in the pericardium; in all the others, the pericardium *adhered* to the heart." The third volume of the "Transactions of a Society for the improvement of Medical and Chirurgical Knowledge," published in the year 1812, contains a paper by Dr. Wells, on "Rheumatism of the Heart," in which he gives the details of sixteen cases where pericarditis arose during the course of rheumatic fever. In this communication, he observes that Dr. Pitcairn, Physician to St. Bartholomew's Hospital, about the year 1788, made the remark, that persons subject to rheumatism were attacked more frequently than others with symptoms of organic disease of the heart. "Subsequent experience having confirmed the truth of this observation, he concluded that these two diseases often depend upon a common cause; and in such instances therefore he called the latter disease "Rheumatism of the Heart." Dr. Latham\* writing in the year 1829, observes: "The disease with which pericarditis is most frequently found in association is acute rheumatism; so frequently in children and young people, that in them our suspicions are always alive to its occurrence." Dr. Elliotson† writing also in the same year, observes: "From the frequency of its [pericarditis] occurrence during acute rheumatism I make it as invariable a rule to examine the cardiac region by touch and hearing in every case of acute rheumatism, as the usual seats of hernia are examined by us all in cases of colic and intestinal inflammation."

If we turn, now, to the French publications on the subject during the period in question, we find no allusion in them to close connexion between rheumatic fever and pericarditis. Laennec makes no mention of it, and M. Louis in his memoir on pericarditis, published 1826, is equally silent on the subject. Indeed M. Bouillaud§ tells us himself (in concluding the detailed case of acute rheumatism, where a post-mortem examination revealed the presence of pericarditis), that in January, 1833, ignorant of the intimate relation between pericarditis and

\* Med. Gaz., Jan., 1829.

† Lumleian Lectures, 182

§ Traité des Mal. du Cœur. t. i., p. 415.

rheumatism. "This case (he says) sufficiently proves that in January, 1833, we ourselves were ignorant of the law of coincidence of inflammation of the heart with violent acute articular rheumatism." "In fact, through the whole course of the disease, the heart had never been examined, and up to the period of the post-mortem examination we had (he says) no suspicion of the pericarditis, which coincided with the articular rheumatism." Nevertheless we find the majority of the modern French writers claiming for M. Bouillaud the credit of having originally traced the connexion between the two diseases; and in the face of the most positive evidence to the contrary, asserting that it had been overlooked by physicians previous to his writing upon the subject. Indeed, M. Gendrin\* goes further, and not only claims for M. Bouillaud all the credit of the discovery, but he accuses Dr. Hope of intentionally overlooking M. Bouillaud's claims.

*Theory of Metastasis.*—When pericarditis arises during the course of rheumatic fever, it is not, as was at one time supposed, the result of metastasis from the joints to the pericardium, but it is to be regarded as an extension of the inflammation from one tissue to another of an analogous nature; in fact, the inflammation in the joints, so far from diminishing on the supervention of pericarditis (which would be the case if metastasis occurred), either remains stationary, or may increase in intensity; while in some instances, the order is reversed, the disease commences as pericarditis, and is followed by articular rheumatism; examples of which have been recorded by Drs. Graves, Hope, Stokes, Watson, Taylor, and the author.

Cases of this kind, showing as they do the intimate connexion between rheumatic fever and pericarditis, go far to prove that the pathological cause of each is the same; and the researches of modern pathologists render it highly probable that this lies in the blood, and that it depends upon the presence of a morbid matter in this fluid (supposed to be either lactic or lithic acid), which has an affinity for certain textures, particularly the fibrous and fibro-serous. Hence the parts which most commonly suffer, as remarked by Dr. Todd † are the joints and the tissues surrounding them, the pericardium, and endocardium; and hence the greater the amount of this materies morbi in the blood, the greater the

\* *Leçons sur les Mal. du Cœur*, 1841–42.

† *On Gout and Rheumatism*.

number of parts which will probably become engaged, and the more intense will be the inflammation. It ought, therefore, to excite no more surprise that the inflammation in acute rheumatism should extend to the investing or lining membrane of the heart, than that it should shift from one joint to another, the structures being analogous.

Although true metastasis from the joints to the pericardium is exceedingly rare in acute rheumatism, it does, nevertheless, sometimes occur. The cases in which it has been observed were all examples of the *capsular* or synovial form of rheumatism, where the synovial membrane is the seat of the inflammation; it has not, that I am aware, been noticed in the fibrous form of rheumatism. Some pathologists deny that metastasis ever occurs; Dr. Walshe ridicules the idea, and calls it an exploded doctrine; Dr. Taylor, however, looked upon its occasional occurrence as both consistent with theory and supported by observation. Indeed we know that true metastasis occurs in gout—a disease which has much analogy with rheumatism; and Mr. Adams\* has recorded two cases where metastasis of erysipelas took place from the head and face to the pericardium; the external redness suddenly disappeared, quickly followed by dyspnoea, agitation of countenance, and orthopnoea. Neither of the patients lived more than two days afterwards, and on examination, the surfaces of the pericardium were coated with recent lymph, and the sac contained a turbid fluid. Mr. Adams, in the same communication, alludes to two similar cases which occurred in the practice of other surgeons.

Every case of rheumatism is not equally liable to be complicated with pericarditis; the age of the patient, the intensity of the fever, the form of the rheumatism, and the tendency the inflammation shows to shift its seat, have each their influence.

*Influence of Age.*—Rheumatic fever is much more liable to be accompanied by endo or pericarditis in young subjects than in advanced life; in fact, in old age it is seldom met with, whereas in early life its non-occurrence is the exception, and the younger the subject the more liable the heart is to be involved. Dr. Fuller attributes this to the “greater irritability of the heart in early life.” Thus, Dr. Watson† says he has known only three persons under puberty to pass through acute rheumatism with

\* Dub. Hosp. Rep., vol. iv.

† Lec. on the Prac. of Med.

untouched heart, and in two of these he was by no means certain that the articular disease was genuine rheumatism. Dr. Durrant\* says "he cannot call to mind a single instance of acute rheumatism occurring under puberty, in which the heart or its pericardial sac was not more or less implicated." Dr. Cargill† says he has not met with a single case of the kind in which the patient "shook off the heart affection and recovered." From the tables given in Dr. Fuller's treatise,‡ it appears that out of 130 cases of recent heart affection arising in the course of acute rheumatism, no less than 114 of the subjects were under thirty years of age. "In the production of exocardial inflammation alone, the influence of age is (he adds) even more apparent; for twenty-three out of forty-one instances, or above one-half, were met with in patients who did not exceed the age of twenty; and thirty-seven out of the forty-one, were under the age of thirty; while four only out of the whole number, exceeded that age." The average age, in sixty-one cases of rheumatic pericarditis, reported by Dr. Ormerod, was twenty-one; while in twenty-four cases of non-rheumatic pericarditis, the average age was forty-two.

*Influence of the Intensity of the Fever.*—The intensity of the fever and the severity of the inflammation in the joints have also an influence upon the frequency of cardiac complication. When the inflammatory fever is high, and the local symptoms are severe, pericarditis is more liable to supervene than under opposite circumstances. M. Bouillaud, Dr. Macleod, and Dr. Budd, all advocate this view; it applies however rather to cases where, in addition to the fever being high, and the local symptoms severe, the inflammation has a tendency to *shift its seat suddenly* from one joint to another. A first attack of rheumatic fever seems to be more frequently accompanied by pericarditis than a subsequent one; this has been accounted for by the first attack being often the most severe. Dr. Taylor says that in fifteen cases of rheumatism, pericarditis supervened in the course of the first attack in eleven; during a second attack in three; and during a third in one. Dr. Budd§ states that out of twenty-six subjects in whom acute rheumatism occurred for the first time, the heart was engaged in sixteen; while out of seventeen subjects, who had had one or more previous

\* Prov. Med. Jour.

† Lond. Med. Gaz.

‡ On Rheumatism.

§ Lib. of Med., vol. v.

attacks of rheumatism, the heart was engaged in five only. The majority of his cases were, however, examples of endocarditis.

*Influence of the Form of the Rheumatism.*—Every form of rheumatism is not equally liable to be complicated with inflammation of the pericardium; it never accompanies muscular rheumatism, neither is it met with in that form which is chronic from the commencement. The capsular or synovial form of rheumatism which is generally sub-acute, is also in a great measure exempt. It seems to be limited almost to the fibrous form of acute rheumatism, or that in which the fibrous tissues of the joints are essentially engaged.

*Stage of Rheumatism at which Pericarditis usually supervenes.*—Pericarditis may set in at any period during the progress of rheumatic fever; it may likewise precede the rheumatism, but this is rare; or both may set in simultaneously, which is still more rare. In general, pericarditis comes on when the inflammation in the joints is at its height, or between the third and the tenth day. In fourteen cases where this point was noticed by Dr. Taylor,\* the pericarditis preceded the rheumatism twice; in two it came on during the first day of the disease; in three on the third day; in one on the fourth; in three on the eighth or ninth day; in two on the eleventh; and in one on the 17th day. Of forty-one cases of pericarditis noted by Dr. Fuller,† nineteen at the date of their admission gave evidence of existing cardiac inflammation; on the average, pericarditis had commenced in these cases before the seventh day. Of the remaining twenty-two, two occurred on the fifth day of the disease; five on the sixth day; four on the seventh; five on the eighth; two on the tenth; two on the twelfth day; and the remaining two on the seventeenth and twenty-fourth days. I have made the remark that, when acute rheumatism commences in the joints of the lower extremities, the heart is most liable to become engaged at the period of its extension to the joints of the upper extremities. It sometimes happens, as I have said, that pericarditis sets in first, and is followed by rheumatic fever; or the patient may pass through the rheumatism without the heart being engaged, and on a relapse endo or pericarditis may occur. In a case of acute rheumatism which was under my care some time since, the heart was not implicated in the first instance; t

\* Med. Chir. Trans., vol. xxviii.

† On Rheumatism.

patient, however, suffered two relapses; in the first of these pericarditis alone; in the second, endocarditis alone ensued.

## FREQUENCY OF PERICARDITIS.

Pericarditis invariably, as M. Louis observes, leaves traces of its existence behind, in the shape of adhesions if the patient recovers, or of lymph, serum, or pus, if he succumbs; hence its frequency can, in some measure, be estimated by the frequency with which these pathological changes are found after death. With the object of arriving at conclusions under this head, M. Louis\* collected all the cases, where an examination of the heart had been made, included in the works of Morgagni, Corvisart, Bayle, Laennec, Bertin, Andral, Rostan, and one or two others, besides all those recorded in four medical journals published in Paris. They amounted to 1263 in number, and 106, or about one-twelfth, presented evidence of the pericardium having been the seat of inflammation. Out of this number, adhesions existed in 70, the remaining 36 laboured under pericarditis at the period of their death. Again, in 443 other cases in which the heart was examined by M. Louis himself, only 18, or 1 in 23, presented traces of pericarditis; of this number, adhesions existed in 11, and 7 were suffering under recent pericarditis. The difference of percentage in the two series of cases, M. Louis considers may be explained by many of those in the first list having been taken from works devoted especially to cardiac pathology, in which a number of examples of disease of this organ are brought together; hence the proportion of cases of pericarditis would necessarily be larger than in a series taken indiscriminately. He concludes from his researches, that the average frequency of pericarditis is about 1 in 23.

Mr. King† has given two statistical tables bearing upon this point, and containing the results of a large number of autopsies. The first includes 665 cases; in 29 of these the pericardium was firmly adherent, and in 20 recent pericarditis existed. The second table includes 944 cases; in 47 of these adhesions of long standing were found; in 30 of them the adhesions were universal; and in 17 partial. He concludes that adhesions occur in 1-23rd of adult subjects. Dr. Taylor found one case of severe pericarditis

\* Mem. Anat. Path.

† The Lancet, Nov., 1845.



in about every eighty of the physicians' cases in University College Hospital. In fatal cases, pericarditis occurred 16 times among 355 subjects, and old adhesions of the pericardium in 1 of every 16 subjects.

A more interesting question, however, than the frequency of pericarditis in comparison with other diseases, is the frequency of its occurrence in rheumatic fever. Dr. Fuller\* gives the following instructive table bearing upon this point :—

	No. of cases of acute rheumatism.			No. complicated with pericarditis.	
Dr. Fuller's cases	...	246	...	...	39
Dr. Basham's do.	...	66	...	...	14.
Dr. Budd's do.	...	43	...	...	5
Dr. Latham's do.	...	136	...	...	22
Dr. Macleod's do.	...	307	...	...	54
Dr. Taylor's do.	...	49	...	...	8
<hr/>					
Total	...	847	...	...	142

Pericarditis was formerly supposed to be the most frequent complication of rheumatic fever ; but statistics prove that endocarditis is much more so. Dr. Fuller's work contains a table of all the cases of acute rheumatism admitted into St. George's Hospital within a given period, amounting to 246 ; out of this number, the heart was affected in 145 instances, of which 114 were examples of recent disease.

Of these 114 cases { 12 were cases of pericarditis.  
75 were cases of endocarditis.  
27 were cases of endo-pericarditis.

Dr. Latham's cases, Dr. Taylor's, and Dr. Budd's, show the relative frequency of the two affections to be nearly the same.

When I come to speak of endocarditis, I shall have occasion to recur to this subject ; for the present, it will be sufficient to observe that pericarditis occurs sufficiently often in the course of rheumatic fever to demand a careful daily examination of the pre-cordial region in every case of the latter disease ; but as to its exact frequency, this probably differs in different localities, and at different seasons ; indeed, during certain seasons in Dublin, the complication is comparatively rare, while in others it is sufficiently frequent. Dr. Copland† is of opinion that pericarditis was

\* On Rheumatism.

† Dict. of Med.



formerly a rarer complication of acute rheumatism than it is at the present day. He says that when he published a dissertation upon rheumatism, many years ago, "his attention was as alive to the circumstance as now, and with equal opportunities of meeting with it in public institutions, and it was much less frequently observed." I believe myself that pericarditis, in former times, was a less frequent complication of rheumatic fever than it is at the present day; and I think this can be accounted for by the mode of treatment usually adopted by the older physicians, consisting in diaphoretics, alkaline and neutral salts, &c., as contrasted with the treatment adopted by their successors. We know now, that alkaline and neutral salts tend not only, in general, to shorten the duration of the disease, but cardiac complication appears to be less frequent when they are had recourse to early.

It was formerly supposed that infants were exempt from pericarditis; but although it is seldom seen in them, cases are on record where the disease occurred at the fourth, fifth, and seventh month. Dr. Lees\* has recorded one where the subject was only four months old; death occurred after long-continued convulsions, and on examination, the surfaces of the pericardium were coated with a thick layer of lymph. In another, communicated to the Surgical Society of Ireland by Dr. Benson,† the infant was but five months old; death took place on the eighth day, and on examination both layers of the pericardium were coated with recent lymph, and some ounces of serum were contained in its sac. Dr. Churchill‡ has recorded another, which occurred in the practice of the late Dr. Hunt of this city, where the infant was seven months old; death took place upon the third day, and on examination the two layers of pericardium were adherent, the lymph being quite recent. The most prominent symptom in the two latter cases was the screaming of the infant.

## COMPLICATIONS OF PERICARDITIS.

We have seen that pericarditis seldom occurs as an idiopathic affection, that in the majority of instances it arises in the course of rheumatic fever, more rarely that it occurs as a sequela, or in states of convalescence from acute diseases. In addition, pericarditis is

\* Pro. of Path. Soc.

‡ Treat. on Dis. of Children.

† Dub. Med. Press.

very often complicated with endocarditis; sometimes with pneumonia, or pleuritis upon the left side, or upon both sides, and sometimes with bronchitis; more rarely with peritonitis, gastroenteritis, or with inflammation of the tissue of the heart. Pericarditis may also supervene upon, or be associated with any disease of the heart, or of the general system. Not unfrequently it supervenes upon enlargement or valvular disease of the heart; it occurs in the course of typhus, or of any of the eruptive fevers; and it is met with in connexion with scurvy, Bright's disease of the kidney, phlebitis, erysipelas, influenza, or delirium tremens. In such cases, however, pericarditis is not the primary disease; and, strictly speaking, they are rather examples of pericarditis complicating other diseases than complications of pericarditis.

The prognosis of pericarditis depends in a great measure upon whether it is complicated or uncomplicated; and if complicated, upon the nature of the complication. Thus, when it arises in the course of rheumatic fever, and the inflammatory exudation consists (as it almost always does if the patient's constitution is good) of plastic lymph, and no other complication exists, it is never fatal in the acute stage, although the disease had been allowed to pursue its course unrestrained, and the patient had undergone no treatment. Even when associated with endocarditis, it seldom proves fatal in this stage. The result, however, is not the same if inflammation of other organs or tissues is combined with the pericarditis; indeed many of those which have been recorded where the symptoms were very urgent and distressing, and the fatal termination rapid, were examples of inflammation of the pericardium complicated with endocarditis and pleuritis, or pleuro-pneumonia.

The idiopathic form of pericarditis is more frequently complicated with pleuritis or pneumonia than the rheumatic form; the inflammation appears to engage almost simultaneously the pleura, or the lung, and the pericardium. In the majority of cases of the rheumatic form of the disease, on the other hand, endocarditis is the only complication, we less frequently meet with either pleuritis or pneumonia, and the prognosis as to the immediate result is more favourable, though the complication with endocarditis often lays the foundation for valvular disease, which tends eventually to shorten life. When pleuritis is associated with pericarditis, that part of the left pleura lining the diaphragm is occasionally alone engaged; in

general, it is limited to the left side, and usually to the portion of this membrane immediately in contact with the pericardium; indeed some writers have attributed the pain of pericarditis to a pleuritic complication. When pleuritis is more extensive, and when fluid is simultaneously effused into the pericardium and pleura, the prognosis is always unfavourable. Peritonitis is a very rare complication of pericarditis, and is scarcely mentioned by writers, though it does occasionally occur. In a case of idiopathic pericarditis, under my care in hospital some time since, inflammation of the pericardium was combined with endocarditis, pleuritis, double pneumonia, bronchitis, and partial peritonitis: such a complication was of necessity quickly fatal.

We are indebted to Dr. Latham\* for some useful statistics bearing upon the complications of pericarditis. Of 90 cases of acute rheumatism in which pericarditis or endocarditis existed, some portion of the lungs, either the pleura, the pulmonary tissue, or the bronchial mucous membrane was inflamed, also, in 19. Pneumonia was the most frequent complication, bronchitis next, and pleuritis was the least frequent. Again, in 24 cases of non-rheumatic pericarditis, given by Dr. Taylor,† pneumonia occurred in 12; while in 16 cases of the rheumatic form, pneumonia occurred in 4 only. In 16 cases of non-rheumatic pericarditis, given by him, pleuritis occurred in 10; and in 24 cases of the rheumatic form it occurred in 7 only.

## DURATION OF PERICARDITIS.

The duration of pericarditis is influenced by a variety of circumstances. The disease may be acute—sometimes it is extremely so; at others, it is sub-acute, or the acute passes into the chronic form. Again, it may be complicated with inflammation of another part, or with diseased conditions of the general system. Finally, the age of the patient, the state of his general health, and the nature of the treatment to which he is submitted, will exercise some influence upon the duration of the disease. In non-rheumatic pericarditis, when copious liquid exudation occurs, or when the inflammation is complicated with pneumonia or pleuritis, or both, its duration may be very short, and it may terminate fatally within a few days. M. Andral has recorded a case which termi-

\* Clin. Lect. on the Heart.

† Med. Chir. Trans., vol. xxviii.

nated fatally in twenty-seven hours; such cases are, however, rare; in the great majority, the average duration is from seven to fourteen days.

When pericarditis sets in during the course of rheumatic fever, and no other complication exists, its duration is usually from eight to twelve days. On the other hand, when it occurs in a subject of broken-down constitution, or in a state of debility from previous illness or other cause, if it does not terminate fatally in the acute stage, it is liable to become chronic and its duration to be protracted.

#### TERMINATIONS OF PERICARDITIS.

Acute pericarditis may terminate:—

1. In resolution.
2. In the deposition of lymph upon the visceral layer of the pericardium, constituting one form of the white patches or milk spots on the pericardium.
3. In partial or complete adhesion between the opposed layers of the pericardium.

The termination in *resolution* is the least frequent, and is observed only when the inflammation had been checked before exudation of lymph occurred, or where lymph in small amount had been effused upon one surface of the pericardium only. It has, however, been asserted that perfect resolution never occurs once lymph is effused; Dr. Latham is of this opinion: "Whenever inflammation of the pericardium has been enough to declare itself by symptoms, the pericardium never (he says) regains its integrity of structure;" "in the most favourable event, the pericardium almost always adheres; medical treatment saves life but it rarely prevents adhesion." Undoubtedly, if the inflammation had been allowed to pursue its course unrestrained, or if treatment was not commenced until the lymph had begun to be organized, we could scarcely expect absorption of it to follow. But if treatment is commenced early, if the amount of exudation is trifling, and if it is deposited upon one surface of the pericardium only, there cannot be a doubt that it may be absorbed, and that resolution may be complete. Indeed, Dr. Taylor has recorded a case where an unequivocal double friction sound had been audible, which disappeared some days before the patient's death; on examination, neither lymph or adh

sions were found, the pericardium was vascular, and contained three or four drachms of serum.

The next most favourable termination of pericarditis is in the effusion of lymph in patches upon the visceral layer of the pericardium, which becomes organized without the opposed surface of the membrane becoming implicated; on examination, one or more opaque white patches are found upon the pericardium covering the right or left ventricle, more rarely the auricles, and not unfrequently at the root of the large vessels, in the form of shreds, or short filaments of organized lymph.

In the majority of cases, the termination is in adhesion. We have seen that the inflammatory exudation consists either of lymph, or of liquid, or of both, and in variable proportions; when the inflammation subsides either spontaneously or as the result of treatment, changes take place in both the liquid and solid portion of the exudation; the liquid portion, if it consists of serum, is quickly removed by the absorbents, but lymph is not absorbed with the same rapidity, or with the same facility; its more fluid part is first taken up, and the opposed surfaces of the pericardium coming in contact, mechanical adhesion takes place between them. The adhesion at this period is slight, and easily broken up; eventually as the lymph becomes organized, the union becomes firmer, and in process of time so intimate that the two layers of the pericardium appear to constitute but a single membrane, and its cavity is obliterated. When the deposition of lymph is partial, the adhesions are partial also, and we find then either threads or filaments, which are adherent by one or both extremities; or thicker and stronger bands, which constitute septa between the layers of the membrane.

General adhesion is much more frequent than partial adhesion. Out of seventy cases collected by Louis, the adhesions were general in sixty, and partial in only ten. In eighty-six cases of old adhesion, referred to by Dr. T. K. Chambers,\* in his *Decennium Pathologicum*, fifty-one were universal, four nearly universal, and twenty-nine partial. When the adhesions are general, and close, the aorta is occasionally found to be diminished in size from its origin, through its whole course in the chest; a point first noticed, I believe, by Dr. Chevers.

The rapidity with which adhesion may take place is remark-

\* Brit. and For. Med. Chir. Rev., vol. xii., 1853.

able, the opposed layers of the pericardium have, in very young subjects, been found adherent throughout, within four days from the commencement of the illness; the lymph in such cases, is of course soft, and the adhesions are easily broken up. When the inflammatory exudation consists in part or principally of liquid, a longer period necessarily elapses; and, if the disease passes into the chronic form, adhesion may never occur.

#### CAUSES OF DEATH AND MORTALITY IN PERICARDITIS.

Rheumatic pericarditis, when uncomplicated, when the subject of it is otherwise healthy, when the disease is recognised early, and efficiently treated from the outset, cannot be said to be a disease which perils life. Idiopathic pericarditis, on the other hand, is a more formidable affection; though this arises rather from the circumstance of its being seldom uncomplicated. Lastly, when pericarditis occurs in a subject whose health is broken down, or when it supervenes in the course of other or serious diseases of the general system, it very generally proves fatal, particularly in the advanced stage of Bright's disease. Dr. Chevers\* says he has never seen or heard of a case of recovery in the latter. "The disease is, in fact, an inflammatory dropsy of the pericardium associated with more or less recent enlargement of the entire heart, and often with endocardial lesions of the gravest character; and even if the fluid could be absorbed, the shreddy lymph with which the serous membrane is abundantly covered, is insusceptible of becoming a bond of adhesion."

M. Louis's statistics show, that out of 106 cases of pericarditis, 70 terminated in adhesion of the pericardium, and in a return to health; while 36 died. In the great majority of the latter, however, the pericarditis was complicated with inflammation of other tissues or organs. Dr. Latham's table contains 7 cases of rheumatic pericarditis, and 11 where endocarditis was combined with pericarditis; of the former none died; of the latter 3 died. Dr. Hope, writing in 1839, says, that during the ten previous years he had not had a single fatal case of pericarditis. Dr. Macleod's table contains 52 cases of rheumatic pericarditis, and only 3 deaths. Finally, M. Bouillaud states the mortality to have been 2 in 14 subjects, treated according to his plan.

\* On Diseases of the Heart, Calcutta, 1851.

When death ensues, either at an early or more remote period in pericarditis, it is more frequently due to the morbid states upon which the pericarditis has supervened, or to the complications, than to the pericardial inflammation itself. The following list of the various morbid conditions of other organs, which may be associated with pericarditis, is from a summary of Dr. Taylor's cases, in the last vol. of "The British and Foreign Medical Review," edited by Dr. Forbes:—

"Acute endocarditis, acute carditis, acute pleuritis, chronic pleuritis, pneumonia, acute bronchitis, meningitis, softening of the brain, hemiplegia, acute peritonitis, erysipelas, phlebitis, inflammation and ulceration of the colon, enlargement of the heart, temporary sanguineous engorgement of the heart, old valvular disease, disease of the coats of the aorta, tubercles in the lungs, emphysema, asthma, œdema of the lungs, serous effusion in the pleura, anæmia, cirrhosis of the liver, atrophy of the liver, enlargement of the spleen, ascites, anasarca, Bright's disease of the kidneys, chronic ulceration of the stomach, and encephaloid cancer of the bladder."

When pericarditis terminates fatally, death may be sudden or gradual; the former is liable to occur if a large amount of liquid is quickly effused into the pericardial sac. The heart's movements being embarrassed by the fluid, and the coronary circulation, more particularly that in the veins, being impeded by the compression which the heart's tissue suffers, the organ may suddenly cease to act, and immediate death ensue. This has generally occurred upon some sudden movement on the part of the patient (as mentioned by Corvisart\*), such as the simple act of rising to stool, or of taking a drink.

When death is gradual, the patient may die from exhaustion; sometimes, when he is apparently progressing to convalescence, a relapse occurs with rapid sinking of the vital powers, preceded occasionally by a convulsion if the patient is under puberty, more rarely by several attacks of convulsions. In other cases, the immediate cause of death appears to be the formation of polypiform concretions in the cavities of the heart, which interfere with the free action of its valves, and impede the circulation through its chambers. In the majority of cases, however, the fatal termination is more immediately due to the complications already alluded to;

\* Avenbrugger on Percussion, translated by Corvisart, p. 408.



and the patient dies from apnæa, or coma, or syncope ensues, from which he never rallies.

#### EFFECTS OF PERICARDIAL ADHESION.

Some difference of opinion exists among pathologists as to the effects of complete and close adhesion of the pericardial layers, 1st, upon the movements of the heart; 2nd, upon its growth and nutrition; 3rd, upon the general health of the patient. Everyone in the habit of making post-mortem examinations must have met with this morbid appearance in subjects who died of very different diseases, and in whom its existence was totally unsuspected.

The older authors looked upon pericardial adhesion as a very grave lesion. The majority of the cases they have related appear, however, to have been complicated with diseases of other parts of the heart, sufficient to give rise to the symptoms mentioned, independent of any adhesion. Corvisart states as the result of his experience, that complete adhesion of the pericardium to the heart will necessarily be accompanied by such derangement of the functions of that organ that death must inevitably ensue, sooner or later, according to age, sex, temperament, profession, &c. Some more recent writers appear to have gone into the opposite extreme. Thus, Laennec\* says he had examined a number of subjects, who never during life made any complaint referred to the organs of circulation or respiration, and who presented none in their last illness, although there was close and complete adhesion of the layers of the pericardium. M. Bouillaud almost repeats Laennec's remarks. Dr. Budd† "has seen a great number of cases of adhesion (often general) of long standing, in which the heart was in all other respects natural, and its functions during life perfectly performed. Finally, according to Dr. Elliotson, pericardial adhesions occasions no greater inconvenience than those adhesions of the pleura so commonly met with; and Mr. W. King entitled a paper communicated by him to the *Lancet*, containing some statistics under this head, "The Harmlessness of Adherent Pericardium."

The experience of most practitioners is, however, opposed to the latter view; Dr. Hope never saw an individual in the enjoyment of good health when complete adhesion existed; there was invariably more or less palpitation and hurried respiration on ex-

\* Auscultation Mediate.

† Library of Medicine.



ertion. In the majority of the cases seen by Dr. Copland, likewise, the functions of respiration and circulation were much disturbed; and Dr. Chevers states, as the result of his experience, that "although general adhesion of the pericardial surfaces, when uncomplicated with valvular disease, often remains for years unattended by any symptom, yet in time it almost invariably conduces to bring about a fatal result, if not by dropsy and failure of the heart, by means equally sure and destructive."

When we consider the nature of the movements which the heart has to perform, such a condition would indeed appear to be incompatible with the perfect accomplishment of its functions; and the subject of it, if he does not suffer from palpitation or dyspœa, must be incapable of making the same exertions as formerly; and, if he belongs to the working class, and his occupation is laborious, it cannot fail of proving a source of distress to him. On the other hand, if his occupation is one not requiring any bodily exertion, it may cause comparatively little inconvenience, unless some acute affection of the lungs or bronchial mucous membrane supervenes. "The chief unfavourable influence which this condition exerts is, probably (as remarked by Dr. Chevers\*), in rendering the right cavities of the heart incapable of adapting themselves to circumstances of temporary and permanent pulmonary obstruction—hampered and embarrassed in their action, their muscular tissue weakened by compression, their dilatability impaired, the safety-valve action of the tricuspid being almost entirely prevented, they become unable to meet that distension which is the first result of nearly every form of pulmonary disease."

#### INFLUENCE OF ADHESION OF THE PERICARDIUM IN GIVING RISE TO HYPERTROPHY, OR ATROPHY OF THE HEART.

From the time of Morgagni to a recent period, it was laid down almost as a general rule, that adhesion of the opposed layers of the pericardium tended to produce hypertrophy of the heart, in consequence of the organ increasing its contractile energy to overcome the obstacle which the adhesion presents to the discharge of its functions; increased action being supposed necessarily to lead to increased nutrition. This opinion is, however, now known to be erroneous, and it has been proved that complete and close ad-

\* Treatise on Diseases of the Heart, Calcutta, 1851.

hesion of the two layers of this membrane not only does not give rise to hypertrophy, but that its tendency is the very reverse, and that it is more likely to be followed by atrophy of the heart. Dr. Chevers\* was the first to advocate this view; "a sufficient number of instances have come under my notice (he says) to prove that where the valves are healthy, complete and close adhesion of the pericardial surfaces has a tendency to be followed by *general diminution* in the size of the heart and its vessels, and by contraction of its cavities." Dr. Barlow† subsequently advocated the same view: "I should, *à priori*, be as little disposed (he says) to anticipate hypertrophy in a heart contracted by pericardial adhesions, as I should be to expect excessive development of the muscles of the leg and foot in a Chinese lady." Mr. W. King's‡ statistical tables tend also to strengthen this view. Recently, Dr. W. T. Gairdner,§ in an excellent practical paper on the subject, has brought forward additional evidence of the correctness of Dr. Chevers' views. In this communication, he has given an abstract of fifteen cases, in which adhesions were found after death in patients who died of various diseases; in the majority, the adhesions were general. In ten of these cases, the heart was not hypertrophoid, or otherwise diseased; in two the heart was enlarged, but the enlargement was accompanied by "considerable valvular or other collateral disease, tending to produce hypertrophy;" and in three the heart was enlarged, but "without valvular or other collateral disease apparently sufficient to account for the enlargement." In two of the latter cases, however, the adhesions were partial.

The adhesive lymph, which constitutes the bond of union between the opposed layers of the pericardium, becomes in the course of time organized, and as in other newly-formed tissues, a process of gradual contraction ensues (something similar to what we observe in the cicatrices of burns), and the thicker the layer of lymph the more considerable will be the subsequent contraction. In the latter case, the muscular tissue of the heart must suffer compression and its diastolic movements particularly must be impeded; on the other hand, when the lymph effused constituted a very thin layer the adhesions resulting present ultimately the appearance of cellular

\* Guy's Hospital Reports, vol. vii.

† Gulstonian Lectures, Med. Gaz.

‡ Ed. Month. Jour. Feb. 1851.

§ The Lancet, Nov. 1845.

tissue, and do not seem to cause much compression of the heart's tissue. In either case, however, the tendency of general adhesion of the pericardial layers is evidently *not* to occasion hypertrophy; although, when either atrophy or hypertrophy are met with after death, it does not follow that either one or the other was necessarily the result of the pericardial adhesion.

For instance, we know, that pericarditis much more frequently complicates acute rheumatism in early life than at any other period; and, if general and close adhesion had formed before the heart attained its full dimensions, and the uniting medium constituted a thick layer, the heart could not afterwards increase in size; and if the subject of it lived to adult age, the organ would be found to be smaller than natural. On the other hand, if pericarditis supervened, and adhesion occurred in a case where the heart at the time was in a state of hypertrophy, the organ, when we come to examine it, would of course be found to be larger than natural, and, as endocarditis is a common complication of pericarditis, and, as pericarditis not unfrequently supervenes upon old valvular disease, there is no difficulty in accounting for the occasional occurrence of hypertrophy, without the necessity of attributing it to the pericardial adhesion.

#### DIAGNOSIS OF PERICARDIAL ADHESION.

The diagnosis of adherent pericardium has always been regarded as difficult, and considerable difference of opinion still exists as to its symptoms. Burns\* looked upon *epigastric pulsation* as a constant symptom; he also notices "a *jarring sensation* in the situation where the apex of the heart ought to beat." Corvisart† mentions, among other signs, "a painful sensation of dragging in the præcordial region; frequent, oppressed breathing upon the slightest motion, syncope, more or less irregularity of the pulse, and absence of strong palpitation, the contractions of the heart being not only quick and disordered, but weak, profound, obscure, and imperfect." Dr. Sanders considered alternate retraction and elevation of the epigastrium immediately below the left false ribs, during the systolic and diastolic movements of the ventricles, to be a positive sign. Dr. Copland, in two or three instances, noticed a pulsation or undulation in the epigastrium, extending sometimes to the intercostal

\* Treatise on the Heart.

† Maladies du Cœur.

spaces of the left false ribs. Dr. Elliotson, on the other hand, has never observed a single symptom, except in one case where a thick adhesion extended along the front of the heart, and the patient had been unable to lie upon the back, on account of a smarting pain in the præcordial region produced by this position.

According to Dr. C. Williams,\* “there is one particular condition in which adhesion of the pericardium occasions an evident sign,” viz., “where in addition to adherent pericardium the pericardial sac adheres to the parities of the chest; in such cases the intercostal spaces are drawn in at each ventricular systole, and the motions of the heart are seen and felt more extensively than natural. The sound or percussion likewise will be always dull at this part, even on a full inspiration, and in every position of the body.” The respiratory movements have also been observed to be less marked than in a healthy state of the parts; and a strong inspiration has not the same effect in lowering the heart. Dr. Law† attaches much importance to this sign, viz., “persistence of the same extent of dulness to percussion in the præcordial region, no matter what position the individual may assume.” The subject of it, he adds, “is quite conscious of the existence of some solid resisting body within his chest, which does not move in the changes of posture of his body, but impedes its motions.” Dr. Law says “he has proved this sign in cases where he has seen the patient all through the attack of pericarditis, and also in cases where the adhesion had been already formed; and he has never found it to disappoint him.”

Dr. Williams‡ has also noticed, in cases of this kind, a peculiar inequality, and intermission of the radial pulse (while the heart's pulsations are quite regular) “some of the pulsations being too weak to reach the wrist, and hence the irregularity.” M. Aran§ regards a diminution in the intensity of the second sound of the heart, or a more or less complete extinction of this sound, as a sign of pericardial adhesion.

Dr. Hope relied upon two signs, which, when combined, enabled him, he states, to diagnose this morbid condition. One was, “that the heart, though enlarged, and when, therefore, it ought to beat preternaturally low down in the chest, beats as high

\* Diseases of the Chest.

† Dub. Quart. Jour., August, 1856.

‡ Lond. Jour. of Med., 1850.

§ Arch. Gen. de Med.

al, and sometimes occasions a prominence of the cartilages of the left ribs." The other (which he regards as more characteristic) is "an abrupt jogging, or tumbling motion of the heart, perceptible in the præcordial region with the stethoscope, however, is not observed unless the walls of the left ventricle are hypertrophied and its cavity dilated. Dr. Durrant\* "want of mobility in the heart itself, when by change of position such can be accurately ascertained to exist, to be a sign of importance." Dr. Taylor† mentions an undulatory movement of the first and second intercostal spaces, and immobility of the heart, when the patient turns upon the right or left side, as was which were present in cases of this kind. Bouillaud has been enabled to recognize pericardial adhesion, in some cases, "by an evident depression, or permanent shrinking in the præcordial region, analogous to that which occurs after a pleurisy, and, "by the embarrassed movements of the heart, perceptible to the hand and ear, the apex not striking the chest wall as of side with its natural freedom." According to Skoda,‡ "adhesion of the pericardium exists, "the apex of the heart moves downwards, and to the left, during the ventricular systole, and upwards and to the right: the apex of the heart does not give a systolic shock, and there is either no shock at all at the apex, or it happens during the diastole." When, in addition, the pericardium is adherent to the sternum and adjacent parts, a sinking in is perceptible sinking in at each systole, in the intercostal space directly in answer to the apex of the heart, or frequently in one or two intercostal spaces higher up." M. Forget§ lays down a list of symptoms which he supposes to characterize pericardial adhesion; it is unnecessary, however, to enumerate them, as they are all common to advanced stages of disease of the heart; and, finally, they are pathognomonic. Finally, Dr. Stokes|| "more than anything else, that there is any certain physical sign of adhesion of the pericardium."

It appears to me that this diversity of opinion, as to the signs which characterize pericardial adhesion, has arisen—  
from not distinguishing the cases where the heart was en-

[ed. Jour., Dec., 1843.

Med. Jour., 1845-46.

Cultivation trans. by Markham.

§ *Maladies du Cœur*, 1851.

|| *On Diseases of the Heart*, p. 21.

larged, from those in which it preserved its normal size, or was atrophied.

2. From not taking into account the morbid conditions of other parts of the heart, which often accompany this lesion, particularly of its valves and orifices.

3. From not distinguishing the cases of complete adhesion from those where the adhesions were partial; or, where the pericardial sac was, in addition, adherent to the pleura, or parietes of the chest.

4. From want of attention to the various alterations of the lungs or pleura, which may complicate pericardial adhesion.

When we consider the intimate nature of the union between the pericardial sac and the cordiform tendon of the diaphragm, it is easy to understand, that, when complete and close adhesion exists between the visceral and parietal layers of this membrane, the apex of the heart will be bound down, and, if the organ is not enlarged, will be prevented from coming in contact with the parietes of the thorax during the ventricular systole, in the normal situation of the heart's impulse. Consequently, no impulse will be felt between the cartilages of the fifth and sixth ribs on the left side, and this part will yield a clearer sound than natural on percussion.

The signs, then, upon which I am disposed to rely, and which I think may be regarded as pathognomonic of complete adhesion, when no complication exists, are—

1. Absence of an impulse in the normal situation, between the cartilages of the fifth and sixth left ribs.

2. An impulse perceptible to the hand and stethoscope higher up than natural, viz., between the cartilages of the third and fourth, or fourth and fifth left ribs, which is communicated by the body of the heart, not by its apex.

3. A clearer sound than natural on percussion over the cartilage of the fifth left rib, and below it in the situation where the heart's apex is felt to beat in the normal state.

4. An undulatory movement in the epigastrium immediately below the xyphoid cartilage; but, as this is present in various other morbid states, it has no value unless associated with the other signs.

When pericardial adhesion is combined with adhesions uniting

the outer surface of the pericardial sac to the parietes of the thorax, we will have, in addition, persistence of the same extent of dulness on percussion in every position of the body, as noticed by Dr. Williams, and Dr. Law; and, at the same time, there may be a retraction, or sinking in, at each ventricular systole, of the intercostal spaces on the left side near the sternum. If the heart is much enlarged, a diffused impulse will be felt over the whole precordial region, but it will not be perceptible much to the left of the nipple or low down, as in hypertrophy with dilatation of the left ventricle; while the heart's action may be irregular, or tumultuous, and confined, "as if one contraction of the ventricle was resolved into a series of short, abrupt contractions, sometimes so feebly and faintly expressed, as if no impulse were communicated to the blood by the heart as it passed through it." If pericardial adhesion is complicated with valvular disease, or with softening of the muscular tissue of the heart, the alterations in its sounds, and in the pulse, noticed by Dr. Williams and M. Aran, may be present. Finally, if the lungs are emphysematous along their anterior margin, or old adhesions unite the pleura on each side over the heart, no sign beyond an undulatory movement in the epigastrium may be present.

If, with the foregoing signs, the patient's history tells, that, at some former period he had been the subject of rheumatic fever, particularly if this occurred in early life, or, if we have had the opportunity of watching the case from the outset, and, the physical signs of pericarditis, after lasting for a certain time, cease, and are succeeded by those which I have mentioned, no doubt can exist as to the presence of adherent pericardium.

#### TREATMENT OF PERICARDITIS.

We have seen that pericarditis arises more frequently in the course of rheumatic fever than under any other circumstances; and, that it is most liable to supervene in cases where the fever is high, the local symptoms are severe, and the inflammation in addition has a tendency to shift its seat suddenly from one joint to another. It is, therefore, a question of much practical interest whether there is any method of treating rheumatic fever capable of preventing the cardiac complication, or of rendering its occurrence less frequent.



Acute rheumatism, there is every reason to believe, depends upon the presence in the blood of an abnormal substance, supposed by Dr. Prout to be lactic acid, by others to be lithic acid; now, if this morbid state of the blood can be corrected, if the *materies morbi* can be eliminated from the system, and its further formation prevented by any particular line of treatment, it is not unreasonable to expect that extension of the inflammation from the joints to the heart may be rendered less frequent. It would, however, be out of place here to enter upon the treatment of acute rheumatism; it will be sufficient to observe that when it has been treated very actively by venæsection and purgatives, or by mercury, cardiac complication is, perhaps, as frequent as when the disease has been left entirely to nature. On the other hand, statistics go to prove that when the treatment by the free administration of alkalies and neutral salts has been adopted (which are supposed to possess the power of eliminating the *materies morbi* from the system), not only is the duration of the rheumatism shortened, but cardiac complication is less frequent. Dr. Furnivall\* who was one of the first to advocate the use of alkalies in acute rheumatism, on scientific grounds, says "I have prescribed alkalies for more than fourteen years in rheumatic cases, of which I must have treated about 400; out of that 400, at least fifty have been cases of severe disease. I have had an opportunity of watching the results of such treatment, and have never failed to use the stethoscope in all such cases, yet in no single instance has heart-disease been caused by the rheumatism when the alkaline treatment had been fairly followed." The preparation which he used was the liquor potassæ, in combination with colchicum. Dr. Basham,† who treats acute rheumatism by large doses of *nitrate of potass* (viz., one, two, or three ounces in two quarts of water, in the twenty-four hours), considers that there is from this method, "a certain amount of exemption from cardiac complication, and cardiac inflammation when present is more amenable to treatment." M. Gendrin‡ also employs *nitrate of potass* (in doses varying from six to twelve drachms in the 24 hours) to the exclusion of other medicines; and it is stated that patients treated upon this plan were less liable to cardiac complication than when any other method was adopted. Dr. Golding Bird has found acute

\* On Diseases of the Heart.

‡ Lond. Med. Gaz., Nov., 1848.

† Proceed. of Med. Chir. Soc., Nov., 1848.



rheumatism yield to no remedy so quickly as the *acetate of potass*, in the dose of half an ounce largely diluted, given in divided doses in the twenty-four hours. The pain diminishes remarkably when the urine becomes alkaline and increases in sp. gr., and he is of opinion that the tendency to affections of the heart is very much lessened after the alkalinity of the urine has been established. Dr. Swett prefers the tartrate of soda and potash, in drachm doses every two or three hours during the day, until the urine becomes alkaline, when it is suspended. Of twenty-five cases treated upon this plan,\* not one was attacked with endo or pericarditis, and the duration of the illness was much less than under ordinary treatment. Dr. Garrod employs the bicarbonate of potash, in the dose of two scruples every two hours, night and day, until the patient has been free from all articular affection and febrile disturbance for two or three days. He has recorded the results of this treatment in fifty-one cases; and he is of opinion that it both shortens the duration of the rheumatism, and prevents or moderates cardiac complication.†

An opinion prevails, and appears to be gaining ground, that rheumatic pericarditis, being merely a local manifestation of the disorder of the general system, similar to that of the joints, the treatment should be directed to the constitutional, rather than to the local affection. The efficacy of mercury in rheumatic pericarditis has also been called in question, because it does not reach the morbid condition of the blood upon which rheumatism depends; and because patients, the subject of acute rheumatism treated by mercury, have been attacked by pericarditis when under its full influence. Undoubtedly, as a general rule, mercury is not followed by the same striking results in rheumatic pericarditis as in some other acute inflammatory affections; and, the majority of the subjects of rheumatic pericarditis recover, although they may not have taken a particle of mercury. The arguments, however, which have been advanced in support of these opinions, do not appear to be conclusive, for the following reasons:—

1st. The local inflammation in rheumatic pericarditis, although it may be but a part of the constitutional disease, is attended with more danger to life than the local inflammation of the joints, and

\* New York Med. Times, Aug., 1854.

† Lancet, March, 1855.

requires therefore prompt treatment specially directed to it, which that in the joints does not.

2nd. Rheumatic inflammation, when involving the pericardium, is not liable to shift its seat, as happens when the fibrous tissue of the joints is engaged; and it is quickly followed by exudation of lymph which is not observed when the joints are the seat of the inflammation.

It is obvious therefore that additional measures are called for, and we should be culpable did we not resort to those which experience has shown to possess the power either of checking inflammatory exudation in serous membranes generally, or of promoting its absorption when effused. Now, none of the remedies, empirically or otherwise employed in acute rheumatism, as the alkalies, colchicum, opium, neutral salts, lemon-juice, nitrate of potass, or quinine, will accomplish the latter objects, however beneficial they may prove when the inflammation is limited to the joints; and we are forced to fall back upon those which experience has proved to be of service in inflammatory affections of serous membranes generally; among which, local bleeding, counter-irritation, and mercury undoubtedly hold the first place.

Whether pericarditis arises idiopathically, or supervenes upon rheumatic fever, the indications to be held in view in its treatment, are—

1st. To diminish fever and local inflammation as speedily as possible.

2nd. To endeavour to prevent inflammatory exudation; but, as this generally cannot be done, to check further exudation, and promote the absorption of that already effused.

3rd. To relieve pain, and the other local symptoms.

4th. To conduct the treatment with as little expenditure as possible of the patient's strength, and to support it all through.

The remedies upon which we especially rely, in order to accomplish these objects, are: 1. Bleeding, rarely generally, frequently locally; 2. Mercury; 3. Counter-irritation, aided by the internal administration of opium, alkalies, colchicum, and other remedies. The earlier that treatment is commenced, the more likely is it to prove effectual; and, as pericarditis is liable to supervene at any time in the course of rheumatic fever, the præcordial region ought, as a general rule, to be daily examined in every case.

has already been observed that acute pericarditis presents under two types, which require corresponding modifications of treatment: in one, antiphlogistic measures must be promptly employed; the other scarcely admits of antiphlogistic treatment, and caution is necessary in its employment.

*Bleeding.*—As in acute inflammation of other serous mem-

branes, bleeding has been extensively employed in acute pericarditis, and under favourable circumstances it has sometimes proved sufficient by itself to subdue the inflammation. Thus if the case

be of idiopathic pericarditis; if the patient is seen at the very beginning and before any exudation has taken place; if his constitution

is sound, if the symptomatic fever is high, and the pulse

is tense, venesection employed so as to produce an impression upon

the circulation, and followed by local bleeding, will sometimes, as

in peritonitis, cut short the inflammation within a few hours. It is

however, in the idiopathic form of pericarditis, when the

disease occurs in a young and vigorous subject, that this *abortive*

treatment, as Dr. Chevers terms it, is likely to prove effective.

A great majority of the cases of acute pericarditis have already

reached the stage which admits of it before we see them; and,

therefore, valuable venesection may be in preventing inflammatory

action, it cannot cause the absorption of the lymph effused as

a result of inflammation.

The most energetic advocate for bleeding in pericarditis is M.

Bouillaud, who trusts almost exclusively to it; and his example

and authority have led to its very general employment on the con-

dition. He advocates not only copious but frequent abstraction of

blood, repeating the venesection several times a day, for two or

three days; together with leeches or cupping to the præcordial

space, and he states with the best results. M. Bouillaud has re-

commended bleeding as early in the disease as possible, but the

presence of lymph or fluid in the pericardial sac does not deter-

mine in all the cases reported in his work the physical signs of

presence are mentioned. M. Bouillaud's practice has, how-

ever, few advocates in this country; bleeding, no matter how

frequently repeated, will not cure pericarditis once the stage of

chronic inflammation has set in, which we know it does early; while the

immediate and remote effects of excessive vascular depletion are

highly prejudicial, by the shock to the nervous system, and

the "embarrassment to the circulation which may arise from converting inordinate into defective action;" as well as by inducing an anæmic or asthenic state of the system, the effect of which will be to render the heart irritable, to protract convalescence, or to give the disease a tendency to pass into the chronic form.

These objections do not, however, apply to a moderate bleeding from the arm, although exudation of lymph has taken place, if inflammatory fever runs high, pain is severe, and the pulse hard; provided it is not employed late in the disease, and the patient's constitution is sound. The venæsection here is not used with the object of cutting short the inflammation, which it cannot do, but with the intention of diminishing fever, moderating vascular action and relieving pain; and whether it produces these effects or not, it will probably have the beneficial one of favouring the absorption of mercury, which as we shall presently see constitutes an important item in the treatment.

*Local Bleeding.*—In the asthenic type of pericarditis, general bleeding is out of the question, and even local bleeding requires caution; indeed, in the great majority of cases even of the sthenic form, which we treat in hospital, we rely principally upon local bleeding. The blood being taken from the immediate vicinity of the inflamed parts, a less amount affords more decided relief than a larger quantity from a vein in the arm, and with less expenditure likewise of the patient's strength. Cupping is preferred by many to leeching, as the blood is more quickly removed and the quantity can be accurately regulated; when, however, the præcordial region is very sensitive, when local bleeding has to be repeated, and generally in the case of females, leeches are preferable. The præcordial region is the best site for local bleeding: Dr. Latham, when he employs cupping, prefers the space between the left scapula and the spine, in consequence of the pain which the cupping glasses occasion sometimes when placed upon the præcordial region.

Dr. Todd\* objects *in toto* to bleeding in rheumatic pericarditis, upon both theoretical, and practical grounds. "It will not, he considers, stop the inflammatory state which creates the undue determination of blood to the pericardial and endocardial surfaces," and it "will not prevent the plastic formations, but rather favour them," while its effects are "to diminish all the solids of the blood

\* Lond. Med. Gaz., Oct. 1848.

it the fibrine, and to augment the water ;" a state favourable to the occurrence of liquid effusion. Experience, he adds, confirms him in the belief " that the practice of bleeding is altogether unsatisfactory in its results, and prejudicial in its consequences; and that the practice of abstaining from this mode of treatment is perfectly safe, and tends to the best results."

*Mercury.*—Simultaneously with the employment of local or general bleeding, or independent of either, mercury constitutes an important item in the treatment, whether the inflammatory exudation consists of lymph alone, or of serum and lymph; and we must endeavour to bring the system under its influence. This may be effected by the administration of calomel in combination with opium, at short intervals, until the gums are affected. If, as not infrequently occurs, an irritable state of the bowels compels us to discontinue calomel, the blue pill or gray powder may be substituted for it; and if there is a difficulty in inducing ptyalism, its constitutional effects may be brought about by applying the ointment externally, either by placing it in the axillæ, or dressing the disordered surfaces with it.

As to the exact dose in which the preparations of mercury should be administered, and the frequency with which they should be repeated, no positive rules can be laid down; we must be guided in each by our knowledge of the patient's constitution, and by various other circumstances to which it is not necessary here to allude. Our objects being to produce the specific effects of mercury early, and to avoid profuse salivation, these will be obtained either by administering this mineral in small doses frequently repeated, or in larger doses and at longer intervals. Dr. Graves\* was in the habit of commencing with a scruple of calomel for an adult of good constitution. Dr. Macleod† considered a scruple in divided doses, within the first twenty-four hours, " to be the average quantity which adults require at the outset of the attack." Small doses frequently repeated are, however, much safer, and equally effectual; it should always be combined with opium, and the addition of a little James' or antimonial powder serves to quicken its specific effects. Should it cause griping or vomiting, each dose may be followed by a draught of the compound chalk mixture, or the compound chalk powder may be combined

\* Clinical Medicine.

† Treatise on Rheumatism.

with it; or the blue pill or grey powder may be substituted for the calomel. As it is of importance that the patient should sleep, a larger dose in combination with a full opiate ought to be given at bed-time.

In persevering in the use of mercury we are principally guided by the auscultatory signs; if the attrition sounds cease within forty-eight hours from the period at which they were first audible, and their cessation is not due to the separation of the surfaces of the pericardium by liquid exudation, the resolution may be complete; but if, as is generally the case, they continue to be audible for several days longer, we cannot expect so favourable a result, and adhesion, which is indicated likewise by the cessation of the attrition sounds, is probable. As a general rule, the mercury is to be continued until the gums are affected, or the attrition sounds cease, when it may be intermitted, given at longer intervals, or in diminished doses, according to circumstances. When endocarditis is combined with pericarditis, mercury requires to be continued for a longer period, and it is generally advisable to persevere in its use in small doses for some days, at least, after the gums are touched.

Dr. Latham considers salivation to be essential; he says, that in two out of the only three fatal cases which he met in a given period, ptyalism could not be induced, although mercury was perseveringly given, and combined with bleeding locally and generally. Dr. Taylor's cases do not, however, show the same favourable results from mercury. It appears, from an analysis of them given by him,\* that salivation was not followed by any speedy abatement of the disease in sixteen cases, while in five it was followed by pericarditis, and in three by an increase in the extent and intensity of the disease. He concludes that "if the production of salivation had anything like the marked influence in arresting inflammation, and in promoting the removal of its products, which it is currently believed to possess, the duration of the cases of pericarditis after salivation ought to have been much less than it really was." Dr. Risdon Bennett, in a recent communication to the Medical Society of London,† related some cases with the object of showing "the non-necessity in a large proportion of cases, and in others the absolute impropriety of attacking rheumatic pericarditis either

\* Lond. Med. Gaz., July, 1849.

† Lancet. 1851.

depletion, whether local or general, or with mercury, so to the system." "He was by no means prepared (he commended the abandonment of mercury altogether, but he was satisfied that in many cases of *acute rheumatism* the disease would not be brought under the full influence of mercury without materially increasing the danger."

Salivation is never necessary, indeed it will prove rather harmful than otherwise; and when renal disease complicates pericarditis, we must trust to other remedies than mercury: "it is not to be caused, it may cause frightful salivation in this form of pericarditis if employed with the utmost care, and with a vivid apprehension of the danger which attends its use."\*

A majority of the French writers appear not to be aware of the antiphlogistic property of mercury, and of its specific effect in the absorption of the exudation poured out by serous membranes when inflamed. M. Bouillaud does not employ mercury in pericarditis, and M. Gendrin† endeavours to prove that it is altogether useless, and that the benefit supposed to have followed its use depended upon the antiphlogistic measures pre-emptively employed.

The hope of preventing the occurrence of pericarditis, or of curing it if it does supervene, in *acute rheumatism* has been vainly bringing the system under the influence of mercury; mercury has no such power, and if given with this object it is injurious rather than otherwise, and we shall be deprived of our most important aids in bringing about the absorption and resolution of inflammation. Dr. Fuller‡ mentions five instances in which acute pericarditis supervened at a time when the patient had profusely salivated, and in every case the ptyalism appeared to operate prejudicially; "the inflammation partook but of the adhesive character, the pericardium became enormously distended with fluid, absorption of this fluid was with difficulty effected, and in two of the cases death was the result."

*Counter-irritation.*—When the acuteness of the inflammation has subsided, and at an earlier period in the asthenic form of the disease, counter-irritation forms a very essential item in the treatment. In some indeed cases are met with where even local bleeding

Chevers on Disease of the Heart.  
Mémoires sur les Maladies du Cœur.

† On Rheumatism.



cannot be borne, and we must depend upon counter-irritation, and internal remedies. The best counter-irritant is the ordinary blister, repeated according to circumstances; it should be of sufficient size to cover the præcordial region, and it may be dressed with mercurial ointment, or followed by an emollient poultice. Blisters, generally, prove most effectual when the exudation consists in part or principally of liquid. Dr. Graves\* says: "before effusion takes place into the pericardial sac, never apply a blister; after it has occurred, repeated and severe blistering over and about the region of the heart is one of our best remedies." In the more chronic forms of the disease, a succession of blisters is preferable to keeping open the blistered surface by irritating applications.

The French practitioners trust principally to antiphlogistics and counter-irritants. M. Guerin employs blisters of large size, frequently repeated; others rely upon digitalis, particularly when the inflammatory effusion consists principally of liquid. But digitalis does not exert its diuretic effects while inflammatory fever exists, and though its specific property may be evinced in cases of dropsical effusion, it has no effect in promoting the absorption of liquid effusion the result of inflammation. Dr. Todd† (who objects to both general and local bleeding in rheumatic pericarditis) employs large blisters, following mustard cataplasms, to the præcordial region; by which a considerable amount of serum, with fibrine, and the white corpuscles of the blood are removed: "by blisters," he says, "you take away that part of the blood which is the great agent in the development of new formations; while that most important part of the blood, the colouring matter, is spared." The only objection to blistering the præcordial region is, that it interferes with a full examination of the part by percussion and auscultation; and, consequently, prevents our judging of the progress of the disease at perhaps a very critical period. In the chronic forms of the disease, where signs of liquid exudation continue after the inflammatory symptoms have been subdued, repeated blistering constitutes the most effectual local treatment.

*Opium.*—After mercury, opium is perhaps one of our most useful agents, by allaying pain, quieting irritability of the heart diminishing restlessness, and procuring sleep. When mercury is admissible, it should be combined with it, and given in a full dose.

\* Clinical Medicine.

† Lond Med. Gaz., 1848.



as two grains at bed-time: when mercury is inadmissible, the opium may be combined with James' or antimonial powder. Dr. Billing\* prefers the salts of morphia, which he considers to be "powerfully antiphlogistic in this disease." "No medicine (he observes) at all equals acetate or muriate of morphia in pericarditis or endocarditis; it allays pain and inflammation, and subdues the pulse, which are the first requisites." "It should be given in frequently-repeated full doses until it conquers the pain, as one-fourth or half a grain every hour; eight or ten grains, (he says,) will be often taken before pain is subdued and sleep obtained." Dr. Fullert† prefers opium to the salts of morphia; he thinks that it exercises some directly curative influence. "In cases of pericarditis the two inflamed surfaces are (he observes) in constant motion, and rub against one another; their innervation is exaggerated—their irritability exalted. Now, although opium cannot of itself prevent the continuance of this friction, it can blunt the sensibility of the inflamed membrane, and make it less obnoxious to the effect of irritation." According to Dr. Corrigan,‡ when acute rheumatism is treated by full doses of opium, the complication of endo or pericarditis is much more rare; among all the cases treated in this way by him, there was but a single instance in which the heart was engaged. His cases are, however, too few to admit of conclusions being drawn from them.

Alkalies, the neutral salts, and the preparations of colchicum are important adjuncts to the other remedies, in the rheumatic form of pericarditis. The former are best given in diluent drinks, the subcarbonates of the alkalies being dissolved in them, or the nitrate of potash may be used instead. They are supposed to prove of use by assisting in the elimination of the rheumatic virus. The preparations of colchicum may be combined with the alkalies, or alkaline carbonates; the tincture of the seeds of colchicum being one of the best preparations, or the tincture of the flowers may be substituted, if the former disagrees. In administering colchicum in these cases, the doses should be small, never exceeding ten drops of the tincture, and if it causes griping or purging it should be discontinued.

When the disease assumes a chronic form, and after the inflam-

\* On Diseases of the Lungs and Heart. † Dub. Jour. of Med., vol. 16, 1840.

† On Rheumatism

mation has been subdued in acute cases, the iodide of potassium in two or three grain doses, repeated several times a day, may be given; it may with advantage be combined with the aqua lauro-cerasi, or tincture of hyoscyamus, or of digitalis; and a good menstruum for it is camphor mixture. This combination will prove of service during convalescence, if the patient suffers from palpitation or want of rest. The acetate or muriate of morphia, or the liquor opii sedativus are also of service at this period, if pains and uneasy sensations about the chest are complained of; and the aromatic spirit of ammonia in camphor mixture, or some light bitter infusion are useful when the patient suffers, in addition, from dyspnoea and palpitation. In some cases, the vegetable or mineral tonics, particularly the preparations of quinine, or iron, answer better; the latter being indicated, more especially, when signs of anæmia are prominent: occasionally, at this period, a Belladonna plaster to the præcordial region will quiet inordinate or irregular action of the heart, when other measures have failed.

Low diet, and the other parts of the antiphlogistic regimen require to be enforced when inflammatory fever is high, but as soon as this has been subdued, and in the asthenic form of the disease from the very outset, the diet must not be spare; whatever nutriment the patient can bear should be permitted, and stimulants or tonics are often likewise necessary. In addition to these remedial measures, rest in the horizontal posture constitutes an essential item in the treatment, and even after convalescence is fully established, excitement of mind, and all unnecessary bodily exertion should be avoided.

#### PARACENTESIS OF THE PERICARDIUM.

In the subacute and chronic forms of pericarditis, after inflammatory action has subsided, but when a considerable amount of liquid remains in the pericardial sac, causing considerable distress to the patient; and, when, instead of diminishing this rather it increases in amount notwithstanding the employment of all the means at our disposal, the operation of paracentesis of the pericardium has in a few instances been performed. I am not aware of any instance in which it has been practised in this country, and but few of its successful issues are upon record.

The first to propose this operation was Riolanus,\* though it

\* Enchirid. Anatom., Paris, 1653. p. 213.

is generally been attributed to Senac. The first to undertake it was Desault;\* his patient died, and upon examination it was found that the pericardium, which was adherent to the heart, had not been opened; a cyst in the pleura containing fluid was mistaken for the distended pericardial sac. Larrey† subsequently performed the operation, but there appears to have been some doubt whether the fluid removed was contained in the pericardium. The operation was performed by M. Schuh, in the Vienna Hospital, upon a patient of M. Skoda's, in the year 1840; the patient died, and upon examination the disease was found to be encephaloid, involving the heart and neighbouring parts. It was also performed, but unsuccessfully, by M. Heger, a pupil of Skoda, in the year 1841; and by M. Béhier, in the year 1854. The three latter cases are given at length in M. Trousseau's memoir,‡ recently published. Laennec speaks encouragingly of this operation when other means have failed, but he never had the opportunity of witnessing it. M. Richerand advocated it, and even suggested injection of the pericardial sac, with the object of bringing about a radical cure by adhesion. Quite recently, M. Aran has carried out M. Richerand's idea; and has shown that an iodine injection may with safety be injected into the pericardial sac.

The first to operate successfully in a case of this kind was M. Romero,§ of Barcelona, who communicated two cases to the Faculty of Medicine, Paris, the details, however, are rather meagre. Subsequently a successful case was reported by M. Karawagen|| of Cronstadt; M. Kyber,¶ however, has had the largest amount of success, four of his patients having recovered. M. Trousseau\*\* has recently recorded another where paracentesis, both of the pericardium and left pleura, was practised in succession with relief to the symptoms; the operation was performed by M. Jobert. When the patient left the hospital, there were evident signs of tubercle in the left lung, with diarrhoea, emaciation, &c. In this memoir he alludes to another successful case in the hands of Dr. Bowditch, of Boston. During the present year, M. Aran brought before the Academy of Medicine, Paris, a case in which he had

\* Œuvres Chirurg. t. ii.

† Mem. de Chirurg. Milit. t. iii. p. 466.

‡ Arch. Gen. de Med. Nov. 1854.

§ Dict. des Sciences Med. t. xl. 1819.

|| Brit. and For. Med. Rev. vol. xii.

¶ Med. Zeit. Russ. & Ed. Month Jour.

\*\* Gaz. des. Hopitaux, Feb. 1855.

twice operated, at an interval of twelve days, removing, in the first instance, twenty-eight ounces of fluid, and in the second forty-nine ounces with relief to the symptoms. On both occasions he injected the cavity with a solution of iodine; that used after the first operation consisted of half an ounce of tincture of iodine fifteen grains of iodide of potassium, and an ounce and a half of water; on the second occasion its strength was increased, and the operation is said to have caused neither pain or uneasiness. The patient had previously manifested symptoms of tubercle, which became more evident as the heart and pericardium improved, and he “finally recovered so far as the affection of the pulmonary organs would allow.”

With respect to the mode of performing this operation, and the exact situation at which the puncture should be made, authors differ. Riolanus originally suggested making an opening in the sternum with the trepan, an inch above the ensiform cartilage, “*si non possis exhaurire istud serum per hydragoga*,” he says, “*licetne terebrâ sternum aperire, intervallo pollicis a cartilagine xiphoide?*” Laennec, without any allusion to Riolanus, suggests the same operation; more recently, M. Skjelderup\* recommended perforating the sternum with the trephine, where the cartilage of the fifth rib joins the sternum, and after the bleeding ceases, when the pericardium protrudes at the aperture, to open it. But this mode of performing the operation is not devoid of danger; the rupture of the mediastinum might, Merat† observes, be followed by the sudden entrance of air into both pleura, the probable effect of which would be the immediate death of the patient.

Desault made the incision “between the sixth and seventh ribs on the left side, opposite the apex of the heart.” Larrey made his incision between the fifth and sixth ribs, “below the nipple.” M. Romero, between the fifth and sixth ribs, at the junction of the cartilage with the bone in men of ordinary stature, and between the fourth and fifth ribs in persons of small stature; he then seized a portion of the pericardium, and incised it with a small curved scissors; the liquid flowed out on placing the patient in a convenient position—a tent was inserted in the wound, and this was removed once a day for three days, and the fluid allowed to escape

\* Acta Nova. Soc. Med. Hav. 1818.

† Dict. des Sciences Med. Art. Pericardite.

ch the edges were brought together, and the wound healed. Wagen performs the operation with a trocar, which he introduces between the fifth and sixth ribs, three fingers breadth from the sternum on the left side. M. Schuh likewise employs the trocar, without any preliminary incision. In the case alluded to, he introduced it between the third and fourth ribs close to the left edge of the sternum, but no fluid following, it was withdrawn and introduced into the intercostal space next lower down. The instrument used by him is known in Germany as "Schuh's trocar;" the instrument is furnished with a stop-cock, to the end of which is fitted a glass tube; this is bent at a right angle, and supports at its extremity a small cup into which the liquid flows, and by its position prevents the sudden reflux of air into the serous cavity. M. Jobert de Lamballe, who operated in the case referred to by M. Trousseau, made his incision in the fifth intercostal space on the left side, one inch from the edge of the sternum down between the intercostal muscles; he then introduced the trocar, covered with a piece of l-beater's skin, obliquely from within outwards. M. Kyber likewise used Schuh's trocar between the fourth and fifth ribs, on the left side close to the sternum. The operation he says is painless, even when it is necessary to remove, by the adaptation of a syringe, blood or air; the entrance of the latter, however, he considers productive of no bad result. "The immediate effects of the operation were return of the pulse, removal of the anxiety and oppression, renewed animal heat, and cheerfulness of mind; at the same time the friction sounds return, and the heart's sounds also again appreciable.†" In the greater number of cases, life is merely prolonged; in four instances, however, recovery followed; and he thinks that if the operation was performed at an earlier period, it would be more frequently successful; "but he thought himself justified in undertaking it, having operated in many cases altogether desperate." M. Aran, in his operation, used a small-sized trocar and canula; which he introduced obliquely upwards in the fifth intercostal space, "a little below where the dulness on percussion was well marked." "The extent of the distended pericardium having been previously ascertained by lines drawn on the chest, and the actual situation of the heart fully ascertained by auscultation."

\* Edinb. Month. Jour., March, 1848.

† Ibid.

If an incision is made between the cartilages of the fourth and fifth ribs on the left side near the sternum, in a subject in which the thoracic organs are sound, we come upon the left pleura, not on the pericardium; and we would be very likely to wound the internal mammary artery, which runs downwards about a finger breadth from the edge of the sternum. The parts divided in the incision, after the skin and cellular tissue, are the origin of the pectoralis major, the expansion of the external intercostal muscle and the internal intercostal, the internal mammary artery lying beneath the latter. The incision, therefore, should not extend to within an inch of the sternum. When the pericardial sac is much distended with fluid, however, it spreads out laterally, pushing the lung and pleura aside, so that the pericardium may be opened between the cartilages of the fourth and fifth, or the fifth and sixth ribs, outside the mammary artery, without any risk of wounding either this vessel or the pleura. The trocar, however, appears to be an unsafe instrument; as, if pushed directly backwards, in the space between the cartilages of the fifth and sixth left ribs, it might wound the diaphragm, and would be very certain to do so between the cartilages of the sixth and seventh ribs, unless the pericardium was much distended, and protruded downwards. Besides, there is a risk of wounding the heart, particularly if bands of adhesion exist between the visceral and parietal layers of the pericardium.

## CHAPTER XII.

## DISEASES OF THE LINING MEMBRANE OF THE HEART.

**ENDOCARDITIS.—ANATOMICAL CHARACTERS OF ACUTE ENDOCARDITIS.—SYMPTOMS.—GENERAL AND LOCAL SYMPTOMS—PHYSICAL SIGNS.—CAUSES AND FREQUENCY OF ENDOCARDITIS.—DIAGNOSIS AND PROGNOSIS OF.—TREATMENT OF ENDOCARDITIS.**

THE membrane which lines the chambers of the heart equally as that which invests its exterior, is often the seat of inflammation, particularly that portion which covers the valves and lines the orifices; indeed, some of the worst forms of valvular disease are the ultimate result of inflammation of this part; and, we are indebted altogether to the researches of modern pathologists for our knowledge of the fact, that inflammation of the endocardium, equally as of the pericardium, is associated generally with acute rheumatism. Inflammation of the lining membrane of the heart was formerly termed "*internal carditis*." M. Bouillaud at first named it "*indocarditis*," to distinguish it from inflammation of the investing membrane; subsequently he altered it to "*endocarditis*;" and, this term has been almost universally adopted by pathologists since.

When upon the subject of pericarditis, I showed that the French writers were greatly in error in asserting, that physicians in these countries were ignorant of the intimate relation between pericarditis and acute articular rheumatism, until M. Bouillaud had pointed out. We are, however, indebted to M. Bouillaud for giving a name to inflammation of the lining membrane of the heart; and, an inconsiderable share of the merit of tracing the connexion between acute rheumatism and endocarditis, belongs also to him.

Every part of the endocardium is not equally liable to inflammation; that portion which covers the valves, or lines the orifices suffers in the majority of cases; the inflammation, too, is generally limited to one side of the heart, or to one cavity; and the valves

and orifices of the left side are much more frequently its seat than those of the right. Indeed, the valves or orifices of the right cavities are seldom alone engaged, or without those upon the left side participating.

#### ANATOMICAL CHARACTERS OF ENDOCARDITIS.

The morbid appearances observed in acute inflammation of the endocardium, are :—

1. Increased vascularity.
2. Dulness, softening, or thickening of the membrane.
3. Exudation of lymph.
4. Adhesions, perforation, or rupture of a valve or tendinous cord.
5. Vegetations upon the valves or orifices.
6. Coagulation of the blood, or fibrinous deposit in the chambers of the heart.

*Stage of increased vascularity.*—As in inflammation in other situations, the lining membrane of the heart, when inflamed, may assume some shade of red; this, however, is not the result of injection of the endocardium itself (which we have seen to be a non-vascular membrane,) but its seat is the subjacent layer of connecting tissue, which is well supplied with blood-vessels; the redness has a pale or rose hue, “the tint being subdued by the endocardium covering it;” or it is spotted, pale and dark, alternately; in one place more of a violet, in another more of a scarlet colour.” We seldom, however, have the opportunity of observing this effect of inflammation, the patient scarcely ever dies at so early a period, and when we come to make an examination, other changes have taken place.

Redness of any shade or hue is not, however, by itself a sign of endocarditis; there are several conditions under which it occurs quite distinct from inflammation. For instance, if the examination was not made until some days after death, or until the putrefactive process had set in, the cavities of the heart being at the same time full of blood in a more or less liquid state; or if a morbid condition of the blood existed, as in persons dying of malignant forms of typhus, small-pox, &c. In such cases, the redness (which is likewise of a darker hue) is due to *cadaveric imbibition*, the colouring matter of the blood giving a permanent stain to the membrane.



Cadaveric imbibation may generally be distinguished from inflammatory redness.

1. By exhibiting no net-work of vessels when examined with is.
2. By being found more frequently at the right than the left of the organ, and extending often into the large vessels.
3. By the darker hue of the redness.
4. By the red colour being more easily removed by maceration.
5. By the endocardium preserving its normal smoothness and polish.
6. By the previous history of the patient's illness.

According to Hasse,\* “the redness of *imbibation* is almost invariably observed in the following descending order; darkest in right auricle, paler in the right ventricle, with the exception of the valves of the pulmonary artery, which are as deeply coloured as the auricle; still paler in the left auricle, while the left ventricle retains quite its natural tint, except that the aortic valves are darker. In the great vessels, the posterior surface is strikingly dark in comparison with the anterior.

*Exudation stage.*—The next effect of inflammation is a diminution of the natural smoothness and polish of the endocardium, accompanied by some softening of the membrane, and by exudation of lymph under or upon the free surface of the inflamed endocardium, which becomes dull and opaque in patches. This may be limited to the valves, or it may extend into the chambers of the heart, and the left auricle is often its seat. When the inflammatory exudation has its seat at the valves, the consequences are much more prejudicial than when it is limited to the lining of the chambers. The effect of swelling, thickening, and puckering of the curtains of the valves, or of shortening of the tendinous cords, of course, is to interfere with the free play of the valves, or to prevent them from fulfilling their office perfectly; and thus either to permit regurgitation, or to obstruct the orifice. This cannot occur without the normal sounds of the heart being modified or altered, or new and abnormal sounds being developed; hence the best signs of endocarditis are generally furnished by auscultation.

*Adhesion of the Valves.*—When the inflammatory exudation has its seat upon the free surface of the endocardium, or reaches

\* Path. Anat. trans. by Swaine.

this part, adhesion of portions of the valves to one another, or to the parietes, may, although it rarely does, take place; this is most common at the arterial orifices, where the valves are thin, particularly the aortic; sometimes the three valves cohere together; more frequently two only cohere, and these are generally, as M. Hasse remarks, the two opposite to the origin of the coronary arteries. "The adhesion commences at the common point of attachment of two valves, and thence spreads to the centre of their free margin, reversing their shape and direction, so that they present a concave surface to the ventricle, and a convex to the aorta. When they preserve their pouch-like character, the orifice of the artery becomes contracted; when, on the contrary, they adhere simultaneously to the parietes of the artery, the mouth of the latter may become permanently dilated." M. Bouillaud says he has never met with adhesion of the arterial valves either to the parietes or to one another; but he has, on a few occasions, seen adhesion of the mitral valve to the parietes, and the posterior lamina was that which was always adherent.

*Perforation or rupture of a Valve.*—It sometimes happens that inflammatory softening ends in perforation or rupture of a valve or of a tendinous cord; this may happen at either the aortic or mitral orifices, more frequently at the former. When rupture takes place, the detached portion of the valve is floated in the direction of the current of blood, and the tendinous cords, when ruptured, eventually shrivel and curl up; both get a coating of lymph, upon which fibrin may be deposited from the blood in its passage through the heart. If the ulceration extends through only one lamina of the curtain of a valve, aneurism of the valve may be the result, and this is more likely to occur if the lesion is situated on that side of the valve against which the blood is propelled. Adhesion of the valves to one another, or to the parietes, or ulceration, or rupture of a valve or tendinous cord, will, of course, either permit regurgitation, or impede the current through the affected orifice, and thus modify or alter the sounds of the heart, or develop new sounds.

*Vegetations upon the valves* are by no means an unusual result of endocarditis; the most common variety consists of little wart-like bodies, about the size of the head of a large pin, composed of lymph, smooth or granular upon the surface, and resembling warts

or the granulations upon an ulcerated surface. Their situation is at the edges of contact and angles of the valves; and, when numerous and seated close to one another, they sometimes form a kind of fringe or beading to it. Occasionally, they are fewer in number and more scattered, sometimes they form a double row, or they spread upon the curtains of the valve, or extend into the interior of the left auricle.

Their usual seat is that side of the valve against which the current of blood is directed, viz., the auricular aspect of the mitral, and the ventricular aspect of the aortic valves. Soft, and easily detached from the endocardium at first, they eventually may disappear, when ulterior changes ensue; or, they become firmer and harder, sometimes acquiring the consistence of cartilage, when they cannot be detached without bringing the endocardium with them. These warty vegetations are seldom met with except in the rheumatic form of endocarditis; and, though most common upon the aortic and mitral valves, they are occasionally found upon the tricuspid: they are least frequent upon the valves of the pulmonary artery.

The other form of vegetation, more rare than the preceding, consists of masses of lymph of larger size and more variable shape, they are also usually fewer in number, and are limited to the left side of the heart; their colour is usually some shade of gray; they are smooth or granular upon the surface, sometimes resembling in colour and appearance the benign polypus of the nose, at others rather the cauliflower excrescence of the uterus. If pedunculated, and according to the length of the peduncle, they hang down into the cavity, and float with the current of the blood; and according to their size, shape, and situation they may obstruct the orifice, impede the action of the valves, or prevent the latter from perfectly closing the aperture.

They are most common in *non-rheumatic* endocarditis; and in a few instances, the endocarditis seems to have been almost perfectly latent. If they occur in the rheumatic form of the disease, it is in feeble, unhealthy subjects, or in individuals who have suffered an attack of acute rheumatism when convalescent from another disease. Both immediately and remotely, they constitute a much more formidable lesion than the warty vegetations. Thus they may be detached, and carried in the current of arterial blood

until arrested in a vessel, the calibre of which does not admit of their passage, as first pointed out by Virchow;\* or the fibrin of the blood in its passage through the heart may be deposited upon them, and then washed away, and carried into the arterial system, until arrested in some part of it. When this occurs, the artery into which the foreign substance is conveyed will be plugged by it, and ultimately obliterated, much in the same way as when a ligature is applied; a remarkable example of which, in one of the arteries of the lower extremity, has been recorded by Mr. Tufnell.† If the supply of blood to an important organ, as the brain, is thus suddenly diminished, serious if not fatal results, as Dr. Kirkes‡ has shown, may ensue. Dr. Todd§ is rather disposed to refer the interruption to the circulation in these cases to a coagulum formed in the artery as the result of arteritis, and connected with a rheumatic or other morbid state of the blood. But, in the majority of cases on record, the coats of the cerebral artery were found to be normal at the part.

Corvisart was of opinion that the warty vegetations had a venereal origin, from their resemblance to venereal warts; this idea is, however, quite exploded now; they are simply a product of inflammation, the shape they assume depending upon the action of a continued current of blood, and, they are found more frequently at the left than at the right side of the heart, because inflammation is much more common there. Dr. Chevers|| considers that the warty vegetations serve a useful purpose in diminishing attrition, and preventing adhesion between opposed surfaces of the valves, as they display no tendency to the adhesive inflammation.

Mr. Simon¶ has recently endeavoured to prove that vegetations are simply deposits of *fibrin* from the blood, in its passage through the heart, which attaches itself to the surface projecting into the current, much in the same way as when recently-drawn blood is stirred with twigs. The vegetations have their seat at the left side of the heart, because arterial blood precipitates its fibrin more readily than venous. But if this were so, these bodies could not become organized, which we know they do. Fibrin mechanically separated from the blood in its passage through the heart, or

\* Archiv. für. path. Anat. vol. i.

† Dub. Quart. Jour.

‡ Med. Chir. Trans. vol. xxxv.

§ Clin. lect. on paralysis.

|| Guy's Hosp. Rep. No. 15.

¶ Lond. Jour. of Med.

through an aneurismal sac, is perfectly *unorganized* and *unorganizable*; it may encrust the lymph which forms these deposits, and so increase their size, or it may form concretions of greater or less size in the cavities of the heart, constituting what we are familiar with as fibrinous polypi of the heart; but, it cannot become organized, any more than blood extravasated from a wounded vessel. In addition, if these vegetations were simply depositions of fibrin, mechanically separated from the blood, they ought to be met with in other acute inflammatory affections, where a similar condition of the blood exists—which they are not; and they would be much more common upon the valves at the right side of the heart than they are known to be.

When exudation of non-plastic matter takes place, or of plastic matter which is not sufficiently adhesive to resist the current of blood, it will be washed off the surface of the valves, and thus mixed with the blood; and as endocarditis is limited generally to the left side of the heart, it will take the course of the arterial blood; but as it is incapable of passing through the capillaries, it will be arrested somewhere in its course, and the spleen and kidneys seem to be most frequently the seat of this arrest.

*Polypiform Concretions in the Chambers of the Heart.*—In any affection in which much impediment exists to the circulation through the heart or lungs, or in which the circulation becomes very feeble, there is a tendency to the separation of fibrin from the other constituents of the blood in the chambers of the heart, constituting the *polypiform concretions* of the heart, which may extend through the orifices, or into the large vessels, obstructing them, impeding the action of the valves, and being sometimes the immediate cause of death. Laennec's "globular vegetations" appear to be only forms of these concretions, and they may occur in endocarditis equally as in other affections in which the circulation through the heart is impeded: some writers, indeed, are of opinion that there is a stronger tendency to their formation in this disease than in others, owing either to the morbid alteration of the blood in endocarditis; or to the inflammation itself determining coagulation of the blood at the affected part, in this respect resembling phlebitis and arteritis, the coagula being more or less adherent in the latter case. Kreysig was the first to advocate the opinion that inflammation of the lining membrane of the heart tends to deter-

mine coagulation of the blood in its cavities: M. Bouillaud takes the same view, and looks upon the development of coagulation as a frequent effect of endocarditis.

*Endocarditis in the Fœtus.*—Rokitansky\* is of opinion that endocarditis is not very unfrequent in the fœtus in utero; the seat then being, according to him, at the right side of the heart, where arrest of development results; by which, obliteration of the foramen ovale or ductus arteriosus is prevented, or contraction of the pulmonary artery brought about. The preternatural conditions which give rise to cyanosis, and which are regarded simply as vices of organization, may, in many instances (he thinks) be the result of inflammation occurring at an early period of intra-uterine life.

Dr. Chevers, in an admirable series of papers, published between the years 1846 and '51,† upon morbid conditions of the pulmonary artery, suggests that the lesions of the right side of the heart, which occur at an early period of intra-uterine life, and give rise to cyanosis, have their cause rather in "malposition of the body of the fœtus," in "pressure upon the umbilical cord," or in "faulty conditions of the lungs." In the great majority of cases, there is no evidence of previous inflammation, it is, therefore, more probable that they are independent of it, and that endocarditis in intra-uterine life plays a very subordinate part in the production of congenital malformations of the heart. Indeed, in a remarkable case, recently recorded by Dr. Mayne,‡ where an infant *four days old* died of endocarditis (the post-mortem appearances being most characteristic), the *left* ventricle alone was the seat of the inflammation; the right cavities being perfectly healthy. Dr. Mayne is of opinion that the endocarditis in this case commenced during intra-uterine life.

#### SYMPTOMS OF ENDOCARDITIS.

The symptoms of acute endocarditis will be considered in the same order as those of pericarditis have been; thus, the symptoms are divided into certain general or constitutional symptoms, certain local symptoms, and certain physical signs. The general symptoms are so similar to those of pericarditis that it is scarcely necessary to do more

\* Path. Anatomy, vol. iv.

‡ Dub. Hosp. Gaz., July, 18

† Lond. Med. Gaz.

refer to what has been said under that head. In the local symptoms there are some points of difference; but the physical signs present the strongest contrast, and it is upon them we especially rely in arriving at a diagnosis.

The general and local symptoms which alone require notice here, are inflammatory fever, palpitation, pain, dyspnoea, and the condition of the pulse.

*Inflammatory Fever.*—Inflammatory or symptomatic fever is common to endocarditis and pericarditis; and the constitutional disturbance will vary according to the amount and intensity of the local inflammation, and the constitutional and other peculiarities of the patient. It is not, however, a sign of much value, as simple, uncomplicated endocarditis is very rare, the disease, in general, arising during the course of acute articular rheumatism, or pleuropneumonia, in which febrile disturbance is well marked. In addition, the symptomatic fever of acute endocarditis does not present any characters by which it can be distinguished from the fever that accompanies acute pericarditis.

*Palpitation.*—Increased or unusual action of the heart, is a frequent and an early symptom of endocarditis; it usually sets in suddenly, and is often the first circumstance which attracts attention to the heart in cases of acute rheumatism. The impulse is stronger than natural, and more diffused, or it may be intermittent, or irregular, at the very outset. The sensation of palpitation is sometimes complained of by the patient, and is distressing to him: at others, nothing of the sort is observed. The palpitation may precede the physical signs, or it may arise simultaneously with them; Dr. Graves\* has made the remark that “an increase of the heart’s action may not only precede the physical but the constitutional signs of inflammation of this organ, or its membranes.” “Connected with the motions of the heart (he adds) is the remarkable disparity that exists between the energy of the heart’s action and the strength of the pulse; for it often happens that the pulsations in the cardiac region are violent, while the pulse is weak and thready at the wrist.” The cause of the palpitation in endocarditis, as in pericarditis, appears to lie in the increased irritability of the muscular tissue of the organ, the result of inflamma-

\* Clinical Medicine, vol. ii.



tion of the membrane in contact with it; it often persists until inflammatory action has been subdued.

*Pain.*—Pain referred to the præcordial region is not so urgent a symptom in endocarditis as in pericarditis, and if the disease is uncomplicated, the sensation complained of is more of oppression or uneasiness, than pain; occasionally no such kind is observed, and the patient denies its existence when questioned. In a few instances, however, the inflammation is attended with severe, burning pain, and strong or irregular action of the heart; in such cases, the endocarditis is always complicated with pericarditis or pleuritis—more rarely with arthritis. Pain may precede the auscultatory signs, or it may not be complained of until the endocardial murmur is fully developed.

*Dyspnœa.*—As long as the circulation through the lungs continues free, dyspnœa is not complained of, the respiration is hurried in proportion as the heart's action is accelerated, and breathing presents nothing peculiar. If, however, the endocarditis is complicated with pericarditis, pneumonia, or pleuritis, or if the circulation through the heart is obstructed or impeded by the effects of the inflammation upon the valves or orifices, dyspnœa becomes an urgent symptom; it generally amounts to a degree of suffocation, and it is accompanied, as M. Bouillaud remarks, "anxious expression of countenance, by perpetual jactitation, an agonizing feeling of suffocation, which prevents the patient from assuming the recumbent posture, and prevents the possibility of sleep."

*Pulse.*—The pulse in endocarditis is usually accelerated, as the disease usually sets in in the course of rheumatic fever; information is to be derived from this character. It has, however, in a few instances that the pulse instead of being accelerated is diminished in frequency on the accession of endocarditis. Taylor\* gives a case where it became not only less frequent, but each individual beat was more slowly performed. In a few cases likewise, there has been no alteration in the pulse of any kind, even in its velocity, throughout the attack. Occasionally the diminution or irregularity of the pulse ushers in the attack; but the variation of the pulse in cases of acute rheumatism, shows

\* Med. Chir. Trans., vol. xxviii.



attention to the heart. Sometimes, the alteration in the pulse precedes the physical signs; more generally it follows them. If the circulation through the heart becomes impeded, although the impulse may be strong, the pulse will be small and weak, or intermittent or irregular, but this state is generally associated with other and more characteristic signs of obstructed circulation.

The general and local signs of endocarditis which have been enumerated resemble in many respects those of pericarditis; in each we have inflammatory fever, and in each we may have unusual action of the heart suddenly ensuing, with pain referred to the præcordial region, and dyspnoea or orthopnoea; without the aid of the physical signs, therefore, it would be impossible to distinguish the one from the other. The only symptom which, as a general rule, presents a contrast is pain, but it is too vague and uncertain a sign to afford much assistance to the diagnosis.

The physical signs of endocarditis are afforded by inspection of the chest, by palpation, by percussion, and auscultation.

*Signs furnished by inspection and palpation.*—Increased or unusual action of the heart is one of the earliest physical signs of endocarditis, and often the first which attracts attention; the impulse is stronger, and more diffused than natural—occasionally irregular. When pericarditis is associated with endocarditis, as often happens, these signs are generally well marked, and the friction fremitus, mentioned under the head of pericarditis, will generally be felt in addition. In the more advanced stages, if the circulation through the heart becomes impeded, the impulse becomes tumultuous or irregular; and if such alteration has taken place in the mitral valve as to permit free regurgitation, a fremitus will be felt, on laying the hand upon the præcordial region.

*Signs furnished by percussion.*—In endocarditis, the region of the heart's superficial dulness is usually increased; the increase however is inconsiderable, at least in comparison with its extent and amount in pericarditis. It is referred by some to distention of the chambers of the heart (particularly the auricles and right ventricle,) by blood; others, with more reason, refer it to the increased extent of the heart's surface which comes in contact with the parietes, owing to increased energy in the action of the organ. Percussion of the præcordial region seldom or never occasions pain in endo-

carditis; whereas, in pericarditis, this manipulation is often painful. The degree of dulness is likewise always more marked in pericarditis, and the space which yields it is wider in every direction; while if much fluid is contained in the pericardial sac, the shape of the dull space will be totally different.

*Signs furnished by Auscultation.*—The effects of inflammation, we have said, are principally expended upon the valves at the left side of the heart, and the changes which ensue as its result may prevent the free play of the valves, or otherwise interfere with their functions; the necessary effect of which is some alteration in the character, duration, or intensity of the sounds; hence, the signs furnished by auscultation are generally the earliest, the most constant, and the most precise.

*Bruit de Soufflet.*—The abnormal sound heard in endocarditis is always some form of blowing murmur, which in the majority of cases replaces or masks the normal first sound of the heart; is generally best marked towards the apex of the organ, and may be so prolonged as to make the second sound appear much shorter than natural. It usually has its cause in insufficiency of the mitral valve; it may however have its seat at the aortic orifice, and be due to an impediment to the outward passage of the blood from the left ventricle; but its most frequent seat in rheumatic endocarditis is at the mitral orifice. This murmur has always more or less of a blowing character, it may pass into a musical murmur, or ultimately into a rough sound; it is described by some as at first a rasping sound, which passes into *bruit de soufflet*; but in all the cases which I have observed, it had the character of *bruit de soufflet* in the first instance.

The exact period which intervenes between the invasion of the inflammation and the development of an endocardial murmur, has not been accurately determined; it is probably as short as that which elapses before the attrition sounds of pericarditis become audible. In general, some alteration is observed in the sounds of the heart from twelve to twenty-four hours before *bruit de soufflet* is developed, the sounds becoming, in some cases, louder or rougher than natural; in others presenting some irregularity of rhythm.

*Triple, or quadruple sounds.*—On several occasions I have observed a doubling of one or other, rarely of both sounds of the

heart previous to *bruit de soufflet* becoming audible. Generally the triple sound is best marked near the apex of the heart, and it gradually passes into *bruit de soufflet*; the doubling has been sometimes in the first, sometimes in the second sound. In a case of endo-pericarditis, recently under my care, a quadruple sound was heard at one part of the præcordial region, and a triple sound at other parts; the triple sound at the base of the heart was produced by a doubling of the first sound, the triple sound at the apex by a doubling of the second sound; the quadruple sound was heard between these two points; the triple sound at the apex passed into *bruit de soufflet*, and the quadruple sound ceased as the inflammation of the endocardium subsided. In this case a *quadruple impulse* was distinctly perceived, on laying the stethoscope upon the part of the præcordial region where the quadruple sound was audible. The cause of the triple and quadruple sounds in endocarditis is evidently want of consonance between the movements of the two ventricles, owing to inflammation engaging the interior of one.

The early appearance of an endocardial murmur has led some pathologists to refer it to "spasm of the valves," or to some other cause, rather than to the changes which supervene as the result of inflammation. But, when we consider, what a trifling degree of thickening, or rigidity of the curtains, or tendinous cords of the mitral valve, will prevent it from perfectly closing the orifice, we can understand how readily a murmur may be developed at this orifice, although no actual change of structure in the valve has taken place.

The valve which primarily and principally suffers in the majority of cases of rheumatic endocarditis is as I have said, the mitral, which becomes incapable of perfectly closing the orifice, and a murmur which obscures or completely masks the normal first sound of the heart, accompanies the systole of the ventricle, the impulse, and the pulse. If the inflammation engages the semilunar valves of the aorta, some impediment may be offered to the outward current of the blood from the ventricle, by which the first sound of the heart would be converted into a murmur; more frequently these valves are rendered incapable of perfectly closing, when regurgitation into the ventricle occurs at each diastole, and a murmur is audible at the period of the second sound of the heart.

It may happen that both set of valves become simultaneously engaged, and a *bruit de soufflet* takes the place of both sounds.

Dr. Barclay's\* statistics of valvular disease, tend to show that both the mitral and aortic valves are more frequently the seat of rheumatic endocarditis than either the mitral singly, or the aortic singly. Thus out of fifteen cases of valvular disease, where it was ascertained that the patient had previously suffered from acute rheumatism, the aortic and mitral valves were both diseased in thirteen, the aortic valves alone in one, and the mitral alone also in one. In another table of thirty-five cases of valvular disease ascribed to rheumatic endocarditis, the aortic and mitral valves were both engaged in twenty-eight, the aortic alone in three, and the mitral alone in four.

But statistics of fatal cases of valvular disease are of little value in determining this point, they merely help to prove what was already well known, that rheumatic endocarditis implicating both set of valves and passing into valvular disease, constitutes a very formidable lesion. So far as I have had the opportunity of judging, rheumatic endocarditis occurring in early life has its seat almost always at the mitral orifice, whereas in non-rheumatic endocarditis, the aortic valves more generally suffer, and both set of valves are not unfrequently engaged.

A difficulty is sometimes experienced in distinguishing the valvular murmur of endocarditis from the attrition sounds of pericarditis. This is scarcely to be wondered at, when we consider that the two affections often arise simultaneously, that a recent attack of endo or pericarditis may be engrafted upon an already diseased heart; that in both diseased states, the heart's action may be similarly deranged or disturbed; and that endo-pericarditis may be further complicated with pleuritis, pneumonia, or pleuro-pneumonia. In the majority of cases, however, the *bruit de soufflet* of endocarditis has positive and well marked characters which distinguish it from the attrition sounds of pericarditis.

*Murmur developed by pressure.*—A source of error may however arise, which must be guarded against, to which attention was first directed by Dr. Latham,† it is, that in children and young subjects, in whom the parietes of the thorax are very yielding, if strong pressure with the stethoscope “sufficient to cause the ribs

\* Med. Chir. Trans., vol. 31.

† Diseases of the Heart. vol. 1, p. 62.

to sink a little below their natural level," is made over the cartilage of the third left rib, a *bruit de soufflet* may be developed, which will cease when the stethoscope is laid lightly on the part. Recently, Dr. Jenner\* has recorded several cases where a *bruit* was developed in this way. The murmur was always systolic, was heard over the base of the heart, and its intensity varied with the degree of pressure. Dr. Jenner is of opinion that this murmur has its seat in the pulmonary artery and is caused by the diminution of the caliber of the artery by the pressure. The conditions favouring its production according to him are, "narrowness of the chest from before backwards, flexibility of the thoracic parietes, and anæmia."

#### CAUSES OF ENDOCARDITIS.

The causes of endocarditis and of pericarditis are much alike, and the two diseases arise under nearly similar circumstances; thus, it is occasionally idiopathic, and is due to the same causes which give rise to acute inflammation of other organs; in general, however, it comes on in the course of acute rheumatism, it is frequently associated with rheumatic pericarditis, and it often accompanies inflammation of the pericardium, arising from other causes; the inflammation appears to extend from the one structure to the other owing to continuity of tissue. We cannot be surprised at this, Dr. Williams† observes, "when we recollect how near the two membranes approach at the auriculo-ventricular orifices, where they are separated by a fibrous structure of more strength than thickness."

Endocarditis not unfrequently arises in the course of Bright's disease, and is sometimes the result of phlebitis. Injuries of the chest would appear also to be an occasional cause; I have met with several cases where it could be traced to an injury of this part. It occasionally occurs as a sequela of the eruptive fevers, particularly scarlatina and measles; it may also follow remittent fever in young subjects. It has likewise been observed as an idiopathic affection in childhood; Dr. West‡ has recorded several such cases. "It does not seem (he says) to be always announced in them by very striking symptoms; a febrile attack of no great in-

\* Med. Times and Gaz. March, 1856.

† Lond. Med. Gaz. August, 1843.

‡ On Diseases of the Chest.

tensity accompanied by increase of the heart's action, are often the only heralds of its approach."

In the following table given by Dr. T. K. Chambers,\* exciting cause of recent endocarditis, in a number of cases, is shown to have been :

				Cases.
Uroemia from Bright's Disease of Kidney	...	...	...	12
Dropsy from Diseased Heart	...	...	...	2
Acute Rheumatic Fever	...	...	...	9
Chronic do.	...	...	...	2
Chorea	...	...	...	1
Do. and Chronic Rheumatism united, &c.	...	...	...	1?
Anæmia, from starvation	...	...	...	1
Typhus	...	...	...	2
Pyæmia	...	...	...	1
Pneumonia (in one case supervening on Phthisis)	...	...	...	3
Pleuritis	...	...	...	2
Malformation of the valves	...	...	...	2
Old disease of the valves	...	...	...	1

#### FREQUENCY OF ENDOCARDITIS.

Idiopathic endocarditis is comparatively rare ; indeed our knowledge of the disease is principally derived from witnessing it as a complication of acute rheumatism. At one time, it was supposed that pericarditis was the ordinary cardiac complication of the latter disease, but statistics show that endocarditis is more frequent, and that when pericarditis occurs, either in connexion with acute rheumatism or independent of it, it is very often combined with endocarditis. Thus, Dr. Latham's work contains a table of 136 cases of acute rheumatism ; out of this number the heart was implicated in ninety, its seat being the endocardium alone in sixty-three, the pericardium alone in seven, and the endo-pericardium in eleven. "It would thus appear (he observes) that two-thirds of those who have acute rheumatism also suffer inflammation of the heart ; and, further, that endocarditis occurs nine times in acute rheumatism for pericarditis once, and that simple endocarditis constitutes more than two-thirds of all rheumatic cardiac affections."

Endocarditis, equally as pericarditis, is more likely to supervene in acute rheumatism when the fever is high, and the local inflammation intense, than under opposite circumstances. M. Bouillaud† says that of 114 cases of acute rheumatism collected by

\* Brit. and For. Med. Chir. Rev. vol. xii.

† Mal. du Cœur, tome ii.

in the space of six or seven years, the inflammatory fever was high in seventy-four, and slight in forty; among the seventy-four, endocarditis or endo-pericarditis occurred sixty-four times; while among the forty, they occurred but once. He concludes that:

1. "When articular rheumatism is acute, violent, and general, the coincidence of endocarditis, of pericarditis, or of endo-pericarditis is the *rule*, and their non-coincidence the *exception*."

2. "In slight, partial, and non-inflammatory rheumatism, their non-coincidence is the *rule*, and their coincidence the *exception*."

Dr. Fuller\* states that between January, 1845, and May, 1848, 379 cases of rheumatism were admitted into St. George's hospital; of these, 246 were examples of the acute form, and 133 of the sub-acute; among the former, recent endocardial affection existed in seventy-five, while it was present in only fourteen of the latter.

M. Bouillaud's and Dr. Latham's estimate of the frequency of endocardial complication with acute rheumatism, is evidently too high. In order that statistics of this kind should be conclusive, it ought to be stated whether any of the subjects had laboured under acute rheumatism previously; whether it had been ascertained that the heart's sounds were normal previous to the attack; and what other signs besides those furnished by auscultation were present in each case; because the number is out of all proportion to what has been observed in other places, and by other practitioners, and without information upon those points, it is difficult to avoid coming to the conclusion that there must have been some source of error, and that in some of the cases at least the recent rheumatic attack had been engrafted upon previous valvular disease. Indeed, Dr. Taylor,\* in reference to this very point, observes: "In a great number, perhaps in most of these cases, the heart disease was of older date than the rheumatism; for in *most* instances in which the physical signs are observed when the patient is first examined, we have no means of distinguishing whether the valvular affection be old or recent." Dr. Fuller is of opinion that "the great excess in the frequency of endocardial affection, exhibited in the tables given by him, arises, *not* from the greater frequency of endocarditis, but from the large number of cases in which a murmur is

\* On Rheumatism.

† Med. Chir. Trans. vol. xxviii.



occasioned either by purely functional causes; or by temporary imperfect closure of the mitral orifice, consequent on irregular contraction of the structures connected with the valves; or by the presence of fibrin deposited on the valves without the concurrence of endocardial inflammation." "This at least (he says) is certain, that the existence of recent valvular murmur was not accompanied by præcordial pain, nor by any local symptoms of inflammation, nor by any increase in the general febrile symptoms in more than 49 out of the 107 cases; and that the murmur ultimately subsided altogether in many of the 58 cases, in which no symptoms of inflammation were observed."

#### DIAGNOSIS OF ENDOCARDITIS.

If it is of importance to detect the early existence of pericarditis, it is of still greater importance that endocarditis should be diagnosed early; the heart, therefore, ought to be carefully examined daily in every case of acute rheumatism, and the slightest alteration in its sounds, anything unusual in its action, or any variation in the pulse should at once attract attention to the organ.

The diagnosis of endocarditis is principally founded upon the auscultatory signs. Dr. Hope\* says it may be anticipated, if a person be *suddenly* attacked with the three following symptoms—1st, fever; 2nd, violent action of the heart; 3rd, a valvular murmur which did not previously exist. The evidence (he adds) is still stronger if the signs occur in connexion with acute rheumatism. Dr. Taylor says he was enabled to diagnose recent endocarditis in a few cases—1st, by the appearance of a *bruit de soufflet* which could not be ascribed to venæsection, or to any other obvious cause, and in a patient who was found to be free from it on admission; 2nd, by some considerable change in the rhythm of the heart, in cases in which it was at first found to be natural."

The diseased states with which endocarditis is most likely to be confounded are:

1. Valvular disease, the result of some previous lesion of the valves.

2. Pericarditis, which very generally coincides with it, and arises under precisely similar circumstances.

\* Treat. on Dis. of the Heart.



3. An anæmic state, the result either of the rheumatism or of the treatment employed.

A difficulty is very likely to arise if the individual at some former period had laboured under rheumatic fever, with cardiac complication. Dr. Taylor, to whose valuable communications I have so often referred, says "he is persuaded that a number of cases of endocarditis cannot be distinguished during life from cases of old disease, because

1. "In old valvular disease, and in recent endocarditis, the modifications in the sounds and impulse of the heart, and in the extent of dulness on percussion are in very many cases the same."

2. "The pyrexia may be a part of the rheumatism, and therefore affords us no aid."

3. "In very many cases of both kinds there is *pain* in the cardiac region, or the pain is plainly seated in the integuments; and in all cases taken singly it is a very uncertain guide."

4. "The permanent disappearance of *bruit de soufflet*, he believes to be the exception instead of the rule in cases of recent endocarditis, and, therefore, it does not generally distinguish this from old valvular disease."

The auscultatory signs of endocarditis have sometimes been mistaken for those of pericarditis; the two diseases are often combined, but the treatment of both being almost the same, an error of diagnosis of this kind would not be attended with material ill consequences. In the majority of cases, however, the *bruit de soufflet* of endocarditis, presents positive and well-marked characters by which it can be distinguished from any attrition sound.

In the following table, the principal points of contrast between the auscultatory signs of each are placed side by side—

CHARACTERS WHICH DISTINGUISH THE BRUIT DE SOUFFLET OF ENDOCARDITIS FROM THE ATTRITION SOUNDS OF PERICARDITIS,

BRUIT DE SOUFFLET OF ENDOCARDITIS.	ATTRITION SOUNDS OF PERICARDITIS.
1. The <i>bruit de soufflet</i> of endocarditis, as a general rule, has a blowing character.	1. The attrition sounds of pericarditis have, as a general rule, a friction character, and present different shades of grating, rubbing, or creaking; very seldom blowing.
2. Usually single and generally accompanying the ventricular systole, the impulse, and the pulse.	2. Usually double, sometimes louder during the diastole than the systole of the ventricles.

3. Generally prolonged, and obscuring or replacing the normal first sound of the heart.

4. Murmur distant and deep-seated.

5. Murmur not limited to the præcordial region; usually loud at and below the apex of the heart, often audible behind at the angle of the scapula or in the course of the aorta.

6. The *bruit de soufflet* of endocarditis preserves the same character throughout, persists in every position of the patient, is tardy in disappearing under treatment, becomes permanent if the disease is left to itself, and often persists after convalescence is established.

3. Generally short; the two sounds follow each other rapidly, and are seldom so loud as quite to obscure the ordinary sounds of the heart,

4. Sounds superficial and near, as if produced at the distal or ear-end of the stethoscope.

5. Almost limited to the præcordial region.

6. The attrition sounds of pericarditis vary in intensity at the same point at short intervals, are sometimes louder in the erect or sitting than the recumbent posture, are usually of short duration, disappear more quickly under treatment, and subside after a definite period, although the disease be left to itself.

A murmur, the result of anæmia, might be mistaken for the murmur of recent endocarditis. Acute rheumatism, we know, has a tendency to induce an anæmic state, owing to the diminution in the red corpuscles of the blood which occurs as its result, and if the disease was treated by depletion and the exhibition of mercury, it is almost sure to follow. Under such circumstances, a systolic murmur is occasionally audible at the aortic orifice, more frequently in the large arteries which arise from the arch of the aorta; and if heard for the first time in a case of acute rheumatism, it might give rise to the suspicion that endocarditis had set in; this would be a serious error, as the treatment of the two states is diametrically opposite.

An anæmic may almost always be distinguished from an endocardial murmur by the following characters.

1. It comes on generally at a more advanced period of the rheumatism; and it is more likely to supervene if bleeding and mercury have been employed.

2. The tone of the murmur is generally soft and blowing, it is often accompanied by a venous murmur in the neck.

3. It is not preceded by disturbance of the heart's action, and the patient usually exhibits other signs of anæmia.

4. It is relieved by tonics, and aggravated by a treatment which would prove of use in acute endocarditis.

## PROGNOSIS IN ENDOCARDITIS.

When endocarditis arises in the course of acute rheumatism, when the patient's constitution is sound, and no complication exists, the prognosis as to the immediate result is always favourable. If the case, too, was seen at its very outset, and treated promptly, we might perhaps prevent the deposition of the products of inflammation, but every day adds to the difficulty, and if the disease was either unrecognized at first, or the patient did not come under observation until some days had elapsed, we can scarcely expect to restore the valves to their original state of integrity. Indeed the acute stage of endocarditis is very short. Dr. Hope says: "after ten days the disease is no longer acute, and the morbid deposit upon the valves has become organized." It is not surprising, therefore, that a *bruit de soufflet* continues to be audible in the majority of cases, after the acute inflammation has subsided, indicating that the valvular lesion, whatever its nature, has become permanent. Thus, Dr. Latham's table, already alluded to, includes sixty-three cases of simple rheumatic endocarditis; out of this number, the endocardium regained its complete integrity in only seventeen, it was permanently injured in the remaining forty-six. Dr. Budd\* goes even further—he says "he has never known the murmur of endocarditis entirely to cease after it was once heard." The influence of the complication of pericarditis with endocarditis, upon the fatality of the latter, is illustrated likewise by Dr. Latham's table. It appears that in sixty-three cases of simple rheumatic endocarditis, there was no death in the acute stage; whereas, in eleven other cases, in which the endo and pericardium were both inflamed, there were three deaths.

The prognosis, as to the remote result, depends in part upon the valve engaged, and upon the nature and amount of injury which it has sustained. Sometimes the lesion is so slight, that with moderate care on the part of the patient, it may neither shorten life, nor materially interfere with his comfort; at others, on the contrary, owing to the seat, the nature, or the amount of the valvular lesion, ulterior changes in the cavities and parietes of the heart ensue, the patient may be rendered incapable of following any arduous occupation, or may become permanently an invalid, and life be considerably abridged.

\* Library of Medicine.

The prognosis, as to the ultimate result, is influenced in no slight degree, likewise, by the age, the habits, the occupation, and the station in life of the patient. As a general rule, the prognosis is much more unfavourable when endocarditis sets in in very early life; few such subjects live to adult age; indeed I have seen it terminate in dropsy, and death, in a young person, within ten months from the period of the attack of rheumatic fever which gave origin to it. The prognosis is more unfavourable in the labouring population, and, consequently, among hospital patients, than in the better classes; the former, who depend for their subsistence upon their daily labour, being often obliged to return to their employment before they are in a fit state to do so, when they are, perhaps, exposed to the same causes which originally produced the disease. If, in addition, the patient's habits are intemperate, the disease is sure to run a more rapid course, and to have a fatal termination, at a much earlier period than under opposite circumstances.

#### TREATMENT OF ENDOCARDITIS.

The treatment of endocarditis is, in many respects, similar to that of pericarditis, but there are also some points of difference; in each, the remedies upon which we especially rely are local blood-letting, mercury, and counter-irritation. The treatment, however, upon the whole, cannot be said to be so satisfactory; we have seldom the satisfaction of pronouncing the patient perfectly restored to health, and the heart to its original state of integrity; some increased impulse continues to be felt, or some abnormal sound continues to be heard, of which, although the patient may be unconscious, yet they too certainly indicate that the valvular apparatus of the heart has sustained injury.

Venæsection, I have said, is capable sometimes of cutting short idiopathic pericarditis, if resorted to at the very outset; not so however in endocarditis, no matter how early it is practised; indeed in every form and at every stage of endocarditis, it is more likely to weaken the patient, than to make an impression upon the disease. M. Bouillaud, however, maintains that frequent and copious bleeding is even more requisite in endo than in pericarditis, but we know that bleeding in acute rheumatism will not prevent the development of endocarditis; it is even considered to favour it, and

perience teaches, that it has not the same power in inflammation of the endocardium, as in inflammation of serous membranes. This objection does not, however, apply to local bleeding, which could be used at as early a period as possible and repeated according to circumstances, though I cannot say, it generally affords the same immediate relief as in pericarditis. Cupping, or leeches to the præcordial region, may be employed according to circumstances; in the cases of females, leeches are to be preferred in this situation.

After local bleeding, our chief resource in endo as in pericarditis is in mercury; but mercury, although the principal agent in causing absorption of inflammatory exudation upon serous surfaces, has much less effect here; the deposit is not readily acted upon by the absorbents, and though often commenced early and perseveringly employed, it too frequently fails in restoring the parts to their pristine state. If, however, we have the opportunity of treating the case from the outset, and mercury is well borne, it should be administered until the system is brought under its influence, when it may be continued in smaller doses and at longer intervals, for some time longer. Dr. Basham\* says, that when acute rheumatism is treated with nitrate of potash in large doses, there seems to be a certain amount of exemption from cardiac complication, and "endocardial, or exocardial inflammation when present, appears to become more manageable and more easily brought under the influence of other remedies when it has been adjunctively employed."

Next to local bleeding and mercury, counter-irritation holds a prominent place in the treatment of endocarditis; the same counter-irritants may be employed here as in pericarditis, and the rules to be attended to in their employment, are nearly similar. The French practitioners are fond of applying digitalis endermically to the præcordial region, by sprinkling the blistered surface with the powder; I have tried it, but the application caused considerable pain, and did not exert the influence upon the heart's action which they represent it to do. Dr. Basham says, that very marked relief has been obtained by covering the præcordial region and anterior surface of the chest with spongio-piline, saturated with nitrate of potash. "In several instances (he says) the irregular murmurs, particularly those of endocardial and pericardial disease,

\* Medico-Chirurgical Transactions, vol. xxxii.

have disappeared even before the system had exhibited any indication of being under the influence of mercury."

Absolute rest is even a more essential auxiliary to the other measures here, than in pericarditis; both body and mind should be maintained in as tranquil a state as possible, and everything calculated to disturb the heart's action should be carefully avoided. The patient, therefore, should be confined to bed during the acute stage, and should be very guarded, for some time after, in using exercise.

The treatment of endocarditis, as a general rule, is more protracted than that of pericarditis; the patient, indeed, cannot be considered safe until the *bruit de soufflet* has disappeared and the action of the heart has returned to its normal state. In too many instances, however, particularly in the subjects treated in hospital, this result is never attained, the patient is compelled to leave the hospital and to return to his employment, he thinks himself well, but finds on resuming his occupation that he is incapable of making the same exertions as formerly, and that palpitation and dyspnoea are more readily excited. Exposure to cold or wet, or intemperance or over exertion, bring a return of his former symptoms and he again seeks admission into hospital, where repose and treatment may so far relieve him that he considers himself well, and he leaves only to return after a longer or shorter period, labouring under anasarca, or other signs of impeded circulation. This is the history of not a few of the cases of valvular disease which we see in hospital and there is a remarkable similarity in the history the patients give of their illness: they had at some former period laboured under rheumatic fever, which was accompanied by palpitation and pain, or oppression in the region of the heart; and their symptoms all date from the period of that attack.

## CHAPTER XIII.

**VALVULAR DISEASE.—CLASSIFICATION OF DISEASES OF THE VALVES.—ANATOMICAL CHARACTERS OF VALVULAR DISEASE.—EFFECTS OF RHEUMATIC AND IDIOPATHIC INFLAMMATION.—EFFECTS OF GOUTY INFLAMMATION.—ADVENTITIOUS DEPOSIT ON THE VALVES.—DILATATION OF THE ORIFICES WITH INSUFFICIENCY OF THE VALVES.—CONGENITAL MALFORMATIONS.—RUPTURE AND ANEURISM OF THE VALVES.**

THE various morbid conditions of the valves and orifices of the heart, included under the general head *Valvular Disease*, are met with at every period of life, and in both sexes, and are occasionally congenital. They sometimes commence insidiously, and are slow in their progress; at others, they set in suddenly, and run their course rapidly. Finally, they are sometimes attended with comparatively little suffering, or they may even exist for a long time unsuspected by the patient; while at others, they constitute some of the most painful and distressing maladies we are called on to treat.

### CLASSIFICATION OF DISEASES OF THE VALVES.

We have seen that the effects of endocarditis are expended principally upon the valves at the left side of the heart, and the foundation of valvular disease is frequently laid in this way. Adventitious deposit is not unfrequent upon the valves, or about the orifices, causing thickening and rigidity of these parts, or obstructing the orifices. It occasionally happens that one of the orifices of the heart becomes dilated without any corresponding increase in size of the valve, which is then necessarily too small to close the aperture perfectly, and regurgitation will be permitted. Again, persons are sometimes born with some imperfection in the valvular apparatus of the heart, the valves may be rudimentary, their curtains may be perforated exhibiting a cribriform appearance, or an irregularity in the number of the valves at the arterial orifices may

under the seven following heads:—

1. Effects of rheumatic or idiopathic inflammation of the heart, and of the changes which the inflammation subsequently undergoes—much more frequent at right side of the heart.

2. Effects of gouty inflammation of the endocardium in a great measure to the left side of the heart.

3. Adventitious deposit upon the valves a common, likewise, at the left side of the organ.

4. Dilatation of the orifices with insufficiency more frequent at the right, than the left side of the heart.

5. Congenital malformation of the valves or to both sides of the heart.

6. Rupture of a valve, of a fleshy column, or of the chordae tendineae, much more frequent at the left, than the right side of the heart.

7. Aneurism of the valves—much more frequent at the left side of the organ.

These several forms of disease, though distinct, may be more or less associated in the same heart, which has suffered from inflammation, may be the seat of adventitious deposit, and, this occurrence may indirectly be the cause of regurgitation at the heart; or congenitally malformed valves may become the seat of inflammation, or become the seat of adventitious deposit.



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THE various morbid conditions of the valves and orifices of the heart, included under the general head *Valvular Disease*, are met with at every period of life, and in both sexes, and are occasionally congenital. They sometimes commence insidiously, and are slow in their progress; at others, they set in suddenly, and run their course rapidly. Finally, they are sometimes attended with comparatively little suffering, or they may even exist for a long time unsuspected by the patient; while at others, they constitute one of the most painful and distressing maladies we are called on to treat.

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exist. In addition, valvular disease sometimes suddenly arises owing to rupture of a healthy valve, or of the tendinous cord, or fleshy column of a valve, upon some sudden, violent muscular exertion. Lastly, saccular or pouch-like dilatations, which get the name of "aneurism of the valves," occasionally form upon their curtains, the result of inflammatory softening, or other cause.

All the varieties of valvular disease met with, may be included under the seven following heads:—

1. Effects of rheumatic or idiopathic inflammation of the endocardium, and of the changes which the inflammatory exudation subsequently undergoes—much more frequent at the left, than the right side of the heart.

2. Effects of gouty inflammation of the endocardium—limited in a great measure to the left side of the heart.

3. Adventitious deposit upon the valves and orifices—most common, likewise, at the left side of the organ.

4. Dilatation of the orifices with insufficiency of the valves—more frequent at the right, than the left side of the heart.

5. Congenital malformation of the valves or orifices—common to both sides of the heart.

6. Rupture of a valve, of a fleshy column, or tendinous cord—much more frequent at the left, than the right side of the heart.

7. Aneurism of the valves—much more frequent, likewise, at the left side of the organ.

These several forms of disease, though distinct in their origin, may be more or less associated in the same heart; thus, a valve which has suffered from inflammation, may become subsequently the seat of adventitious deposit, and, this occurring at the left side, may indirectly be the cause of regurgitation at the right side of the heart; or congenitally malformed valves may suffer from inflammation, or become the seat of adventitious deposit. Disease is usually limited either to a single valve or orifice, or to one side of the heart; and is by many degrees more frequent at the left than at the right side of the organ.

#### EFFECTS OF RHEUMATIC OR IDIOPATHIC ENDOCARDITIS.

Acute inflammation of the endocardium covering the valves and lining the orifices of the heart, occupies a conspicuous place in the production of valvular disease, but not to the extent that

Some writers would lead us to suppose; many of the worst forms of valvular disease undoubtedly have their origin in it, though this is due as much, perhaps, to the changes which the inflammatory deposit subsequently undergoes; but valvular imperfection, as we shall presently see, is not unfrequently entirely independent of inflammation.

Under the head Endocarditis, the morbid changes which have their origin in inflammation were described; and we found that lymph may be deposited either *upon* the membrane which gives a covering to the valves, and lines the cavities of the heart, or *between* the laminae of the valves. The inflammatory exudation does not, however, remain stationary; ulterior changes ensue in it, or a fresh attack of endocarditis leads to additional deposit. The latter is most liable to occur in cases of rheumatic endocarditis—a previous attack of acute rheumatism complicated with cardiac inflammation appearing, as Dr. Barclay\* remarks, to confer a predisposition to suffer from subsequent endocarditis, even though the rheumatic disease should not again manifest itself in the joints.

When the inflammatory exudation has its seat between the laminae of the valve, more or less opacity, thickening, and rigidity of the valve are the necessary result; it acquires a dull, opaque appearance, as if it had been macerated, its tissue becomes more dense, and its elasticity is impaired. The exudation matter, in process of time, becomes converted into fibrous or fibroid tissue, which, contracting and shrivelling, occasions shortening and puckering of the valve, or of its tendinous cords; and in the end, it not unfrequently becomes the seat of atheromatous, or calcareous deposit.

When the *aortic* valves are thus diseased, they can no longer meet to close the orifice, which remains patulous, and permits regurgitation at each ventricular diastole; or the rigid and thickened valves, in addition, impede the outward current from the ventricle; eventually they become the seat of adventitious deposit, and their valvular functions are permanently impaired. This morbid condition of the semilunar valves, was first particularly described by Dr. Hodgkin,† under the name “*retroversion of the valves of the aorta* ;” he noticed the double murmur which sometimes ac-

\* Med. Chir. Trans., vol. xxxi.

† Lond. Med. Gaz. vol. iii, 1829.

they may render the surface over which the blood flows rough and uneven, and thus give rise to a murmur at the orifice. The latter effect is more common at the aortic, the former at the mitral orifice. These warty vegetations, according to Dr. Ormerod, do not constitute a permanent lesion, they disappear in the course of time, and the process by which this is effected has been accurately described by him.\* I have, however, in two instances, found these bodies upon the margin of the mitral valve, although the attack of rheumatic endocarditis which gave origin to them had occurred at least fifteen years previously.

*The larger vegetations* (which are the result of non-rheumatic endocarditis, or if they occur in the latter, it is in subjects whose health is broken down) constitute a much more formidable lesion. They are not to be confounded with Laennec's "*globular vegetations*," which appear to be only a form of fibrinous concretion. Their usual seat is on the aortic valves, their colour is yellowish-white or some shade of grey, though sometimes red from imbibition; their size, shape, and number vary, they may be solitary or numerous, their surface is rough and granular, or smooth, they may be lobulated or pedunculated. "When of recent origin they are soft, and easily lacerable; when of longer duration, they are hard, sometimes cartilaginous, or even penetrated with calcareous matter;" and, according to their size, shape, or number they may obstruct the orifice, or prevent the closure of the valves. In a few instances, however, they have served a really useful purpose by preventing regurgitation, as where one of the aortic valves is partially destroyed by ulceration, they may, by filling up the aperture, act in some measure as a valve, during the ventricular diastole, and prevent regurgitation; a well-marked example of which was recently presented by a patient under my care. When these large vegetations are pedunculated, they hang down into the cavity, and are floated backwards and forwards with the current of the blood. If their attachment is slight, they may be separated, or break off, when they will be carried in the direction of the arterial current until arrested in some vessel the caliber of which is insufficient to admit of their passage; and according to the situation of this vessel, or the importance of the parts which it supplies, a different train of symptoms will supervene.

\* Gulstonian Lectures, Med. Gaz.

*Adhesion, Perforation, and Ulceration.*—Another but a rarer effect of endocarditis is adhesion of the curtains of the valves to one another. At the aortic orifice, if this happen, the margins of the valves may be united to one another, leaving a narrow aperture in the centre, through which the blood is with difficulty propelled. Or, the curtain of one of the valves may contract adhesion with the walls of the aorta; and if this happens to the valve opposite the origin of one of the coronary arteries, it may, in addition to causing permanent patency, obstruct the orifice of the coronary artery, and thus, by interfering with the proper nutrition of the tissue of the heart, lead to degeneration of its muscular fibre. Adhesion is more frequent in the curtains of the mitral valve; and, its effects here are not only to obstruct the orifice, but to prevent the valve from perfectly closing the orifice.

Ulceration and perforation of the curtains of a valve are occasional results of endocarditis; ulceration is most frequent at the aortic orifice; we occasionally find one of the semilunar valves broken down, and presenting jagged and irregular edges, evidently the result of inflammatory softening; or, the curtain of one of these valves may be perforated, or the ulceration may extend through only one lamina, and aneurismal dilatation of the valve be the result. The affected valve may ultimately become the seat of adventitious deposit, or of vegetations, or fibrinous deposit may form upon it. Appearances closely resembling the latter effects of inflammation, may be the result simply of atheromatous and calcareous deposition upon these valves, and of the changes which such deposit brings about.

#### EFFECTS OF GOUTY ENDOCARDITIS.

The morbid appearances which have been now described, are the ordinary results of rheumatic or idiopathic endocarditis; the effects of gouty inflammation of the endocardium differ in several respects, and are sufficiently characteristic, although but very cursorily alluded to in systematic treatises; in fact, Dr. Wardrop\* is almost the only author who has treated at large on the subject, and we are indebted to him for much of the information we possess respecting it.

“Reasoning from analogy (Dr. Wardrop observes), and bear-

\* Treatise on Diseases of the Heart, 1851.

ing in mind the natural structure of both the pericardium and endocardium, we are led to anticipate that when gout affects the heart the inflammation will be seated not in its muscular structure, but in either of these serous membranes. When the eye is attacked with gouty inflammation, the serous membrane which lines the cornea, spreads over the surface of the iris, and extends over the anterior surface of the capsule of the lens, becomes dull and discoloured from effused lymph; and if the inflammation has been severe and of long duration, or if the attacks have been frequent, portions of the capsules of the aqueous humour and lens become thickened, and in some instances are even ossified. Now these morbid changes are precisely those of *arthritic endocarditis* and *pericarditis*; the lining membrane of the heart loses its transparency and polished surface, its various portions, such as those reflected upon the valves, become thickened with a cretaceous or osseous matter deposited between its laminae.\*

The earliest effect of gouty inflammation is loss of transparency in the endocardium covering the valves, which often extends to the membrane in the vicinity, and is accompanied by slight thickening of the valves. In a more advanced degree, the endocardium covering the valves is not only opaque, but the latter are evidently thickened, owing to deposition under the lining membrane, or increase of thickness of the basement membrane. The opacity extends to the endocardium in the vicinity of the valves, and may either spread over their curtains, or form patches of variable size. Eventually the valves become rigid or puckered in addition, and calcareous deposit to a greater or less extent occurs upon them.

These morbid appearances are rare at the right side of the heart, and are almost limited to adult age, and advanced life. They are met with in both sexes, but are more common in the male; they affect both the aortic and mitral valves, but the former most frequently. The appearances presented by the lining membrane in the earlier stages are sometimes attributed to advanced age, at which period there is a predisposition in transparent tissues to become opaque; or, they are described under the head of "hypertrophy of the fibrous tissue of the valves;" but there can be little doubt of their gouty origin in most cases.

The changes which ensue as the result of gouty inflammation

\* Treatise on Diseases of the Heart, p. 495.

the endocardium are never marked by the prominent symptoms which characterize acute rheumatic or idiopathic endocarditis; indeed the affection is often latent, and always chronic and intermittent; and, it does not give rise to symptoms of obstructed circulation unless the valves have become rigid, contracted, and thickened, or calcareous deposit has taken place on them; and even then, the symptoms are much milder than in a similar condition of the valves, the result of some other causes.

#### ATHEROMATOUS AND CALCAREOUS DEPOSITS.

The adventitious deposit which gets the name of *atheroma* is common to the large arteries and the valves of the heart, is much more frequent in advanced than in early life, and at the left than the right side of the organ. The changes which ensue as the result of this deposit are essentially of a chronic nature, and totally independent of inflammation.

Atheroma makes its appearance in the form of "small, opaque, white, or buff-coloured spots, either isolated or grouped together in irregular patches;" it is scarcely elevated at first, and its seat is beneath the lining membrane, to the under surface of which it adheres slightly; so that if the endocardium at the part be raised, the opaque patch comes away with it. The spots increase gradually in size, form elevations upon the surface, coalesce, and eventually constitute patches of variable size and irregular shape. Atheroma belongs to the class of fatty deposits, and was shown by Mr. Gulliver to be composed of oil-globules, cholesterine, and granular matter. The fat or oil-globules were first detected in it by Gluge in 1839; subsequently, this deposit was very fully described by Mr. Gulliver.\* The seat of atheroma is always under the endocardium; at the aortic orifice it is met with, as Bisot remarks, at the base of the semilunar valves, before it is observed upon their curtains; and it engages that surface of the valves which looks towards the aorta, rather than the ventricular aspect.

The changes which atheromatous deposit undergoes are not altogether the same in the valves and the large arteries; in the latter it softens, when it sometimes gets the name *steatoma*, or fat scales of calcareous matter are deposited in it. At the valves,

\* Med. Chir. Trans., vol. xxvi.



softening is less common ; calcareous deposit, however, is frequent, but this occurs generally in irregular nodules or masses.

*Calcareous Deposit.*—Calcareous deposit, or cretification as it is sometimes called, is most frequent and most abundant about the semilunar valves of the aorta, but is not uncommon upon the mitral valve. This deposit may occur as a sequel to atheroma, or independent of it ; in the latter case, as Dr. Wardrop\* has shown, it is frequently the result of *gouty* inflammation of the endocardium. The deposit is sometimes friable, sometimes extremely hard ; it “ consists essentially of carbonate and phosphate of lime ;” it presents itself in different degrees, and assumes a variety of forms ; the corpora Arantii are sometimes enlarged by it ; at others the margin of the thickened valves or their laminæ present irregular, angular, or jagged nodules or prominences. When partial and slight, the valves may still perform their functions ; but when extensively deposited, and the indurated valves coalesce at their margin, the aperture assumes a triangular shape, or is a mere chink through which the blood is propelled with difficulty, and through which, likewise, regurgitation is permitted. At the same time the orifices of the coronary arteries are sometimes obstructed, or contracted by the deposit.

When calcareous deposit occurs in large patches at the base of the semilunar valves, the curtain of one is sometimes united by it to the parietes of the aorta ; or, one of the rigid valves stands out from the others, the orifice is imperfectly closed, and regurgitation permitted. In other instances, interstitial absorption goes on until the curtain of the valve is so much thinned, that perforation or rupture occurs on a comparatively trifling muscular exertion. Perforation or rupture of a valve produced in this way is liable to be mistaken for the effects of inflammatory softening or ulceration ; indeed, as Dr. Chevers remarks, “ unless the injury has been very recent, it is extremely difficult to assign its real date or cause from a mere inspection of the parts.”

At the mitral orifice this deposit may occupy the margin of the lamina of the valve, particularly its ventricular aspect ; more rarely the tendinous cords. The calcareous matter may be deposited in nodules or masses, occasionally in scales. In extreme cases this valve becomes indurated, rigid, and inflexible, and the

\* On Diseases of the Heart.



culo-ventricular aperture is contracted, offering a high degree mechanical impediment to the entrance of the blood from the left ventricle, and at the same time imperfectly closing the aperture. When the deposit occupies the tendinous cords of this valve, they become contracted, and are rendered brittle, and liable to rupture from comparatively trifling causes.

The frequency of adventitious deposit at the left side of the heart, in comparison with the right, the influence of age in favour of the left, and its connexion with the same deposit in the aorta at its base, are shown by all the statistical tables which have been published. M. Bizot met with spots of atheroma upon the aortic valves in 50 out of 156 subjects who died of other than cardiac disease, and ossific deposit, besides, in thirteen of those; the pulmonary valves were not implicated in a single instance. Dr. T. Chambers,\* in his *Decennium Pathologicum*, states that chronic disease of the valves was met with in 367 out of 2161 bodies examined. The valves were thickened or contracted in 156, and contained chronic morbid deposit in 211 of these cases.

‘Of the 156 cases of thickened or contracted valves,

				Times.
The Mitral and Aortic were affected simultaneously				50
The Aortic alone	...	...	...	48
The Mitral alone	...	...	...	37
The Mitral and Tricuspid		...	...	8
The Mitral, Aortic, and Tricuspid		...	...	5
All Four Sets	...	...	...	4
The Tricuspid alone	...	...	...	1
The Tricuspid and Aortic		...	...	2
The Aortic and Pulmonary		...	...	1

‘Of the 211 cases with chronic morbid deposits,

The Mitral and Aortic were affected simultaneously				71
The Mitral alone	...	...	...	59
The Aortic alone	...	...	...	59
The Aortic, Mitral, and Tricuspid		...	...	6
All Four Sets	...	...	...	5
The Mitral and Tricuspid		...	...	2
The Aortic, Mitral, and Pulmonary		...	...	2
The Aortic and Pulmonary		...	...	2

In the 156 cases of thickened valves, atheroma of the aorta

\* Brit. and For. Med. Chir. Rev. vol. xii. 1853.

was noticed 62 times. In the 211 cases of chronic deposit, the atheroma of the aorta was noticed 90 times.

Atheroma and calcareous matter are not the only morbid deposits met with upon the valves; we sometimes find the fleshy columns of the auriculo-ventricular valves infiltrated with fat, or converted in a great measure into adipose matter, predisposing to rupture of these parts. In such cases, however, more or less of the muscular tissue of the heart has undergone the same morbid change, and it will come under consideration in the chapter on fatty degeneration.

#### DILATATION OF THE ORIFICES WITH INSUFFICIENCY OF THE VALVES.

The physical signs of valvular disease are sometimes present, although there is no actual disease of the valves; and regurgitation may take place independent of any morbid condition of these parts. This may occur at either of the auriculo-ventricular orifices, or at the aortic; it is most common at the tricuspid, and most rare at the pulmonic orifice. For instance, when the right auricle and right ventricle become dilated from any cause, the auriculo-ventricular orifice usually also becomes enlarged, and the tricuspid valve remaining stationary, is then incapable of closing the orifice, and regurgitation is permitted; when the phenomenon with which we are familiar as *jugular pulsation* will be observed.

It sometimes happens that dilatation of the *left* auricle and ventricle is accompanied by increase in size of the auriculo-ventricular orifice on the same side, in consequence of which the mitral valve is incapable of perfectly fulfilling its function, and regurgitation is permitted. This, which is very common at the right side of the heart, is comparatively rare at the left, and when regurgitation does occur in this orifice, it is, in the great majority of cases, the result of disease of the mitral valve itself.

Dilatation of the ascending portion of the arch of the aorta is by no means rare, and the aortic orifice not unfrequently participates in the dilatation, or the latter may occur independent of it; nevertheless, regurgitation does not often result from this cause alone, because the tissue of the valves yields and expands, they become increased in all their dimensions as the orifice becomes dilated ("the elastic tissue of which they are composed being capable of undergoing great extension under pressure gradually

plied,") and they are thus enabled to fulfil their valvular function perfectly.

When the *aortic* orifice is dilated, and the semilunar valves are increased in size, retroversion of one of them may ensue upon sudden or unusual muscular exertion; this does not, however, often happen, more frequently the valves become sacculated below. If, however, the valves in the first instance were thickened, or otherwise altered by disease, so as to be incapable of yielding or expanding, and the aortic orifice then became dilated, they would be incapable of closing it, and regurgitation into the ventricle would ensue as a necessary result.

In cases of congenital malformation of the heart, the orifice of the *pulmonary* artery is occasionally dilated, rendering the valves insufficient; a very interesting example of which, where the physical signs were noted with great care, has been recorded by Dr Gordon. "In cases of old bronchitis and extensive emphysema of the lungs, as well as where death has resulted from mitral and aortic obstruction, it is usual (Dr. Chevers\* says) to find the pulmonary artery with its valves, more or less, sometimes extremely dilated, coarse in structure, and irregularly thickened and opaque." In extreme cases, "the main trunk and valvular apparatus of the artery are found to have undergone, in addition to their dilatation, a marked degree of thickening, and to have become the seat of a certain amount of opaque interstitial deposit." He has, however, never met with a case where the pulmonary valves had become inefficient in consequence of retroversion, or from any other causes than mechanical violence, or congenital defect; and only a few cases of the kind are upon record, while in these, some of the other orifices of the heart were likewise dilated. One of the most remarkable cases of the latter class is recorded by Dr. Stokes;† in this, all the cavities of the heart, the orifices of the aorta and pulmonary artery, and both auriculo-ventricular orifices were dilated.

#### CONGENITAL MALFORMATION OF THE VALVES.

Congenital malformation is met with at both sides of the heart, but most commonly at the right; and it may engage the arterial or auriculo-ventricular valves, more frequently the former. Its

\* Lond. Med. Gaz.

† Treat. on Dis. of the Heart.

effects are generally to render the valves incapable of fulfilling their functions, seldom to obstruct the orifice. Congenital malformation of the valves may be all included under the four following heads:—

1st. Cases in which a cribriform condition of the curtains of the valves exists.

2nd. Cases in which one curtain of a valve is too small, or a tendinous cord too short to allow of the perfect approximation of the edges of the valve; a condition originally described under the name *atrophy of the valves*.

3rd. Cases in which the valves are rudimentary, or there is some irregularity in the number of the signoid, or semilunar valves.

4th. Cases in which one of the arterial orifices is contracted, the result of disease during intra-uterine life.

*A cribriform condition of the valves* is the most common congenital malformation of these parts, and is observed more frequently at the arterial than at the auriculo-ventricular orifices. The apertures may be single or several, in general there is more than one; they vary in size, being sometimes no larger than pin-holes, sometimes sufficient to admit a quill; their shape when very small is circular, when larger elliptic or oval; in a few instances they were so large and numerous as to give the part a reticulated appearance. They may occupy but a single curtain of one valve, or more valves than one in the same subject; they are met with in both sexes, and are nearly equally frequent at the aortic and pulmonary orifices. M. Bizot says that in 157 subjects of both sexes, he met with this condition 58 times in the semilunar valves of the aorta, and 51 times in the pulmonary valves (but there is probably some mistake in the numbers given by him); he does not appear to have ever seen it at the auriculo-ventricular orifices. Rokitansky\* says it is only found at the arterial orifices, and more especially at the aortic; Dr. Kingston,† however, met it twice in the tricuspid valve, and once in the mitral; I have seen it in the mitral valve, where it gave rise to well-marked physical signs; several apertures existed, some of which would admit a small quill.

This cribriform condition of the valves is attributed by some writers to atrophy, by others to disease (ulceration), but the other

\* Man. of Path. Anat. vol. iv.

† Med. Chir. Trans. vol. xl.

parts of the valve present no abnormal appearance, except that the curtains in some instances are somewhat thinner than natural; the edges of the aperture are perfectly smooth, are lined by endocardium, and exhibit no traces of inflammation. There can be no doubt, therefore, that it is congenital, and it probably has its cause, as Dr. Chevers\* observes, in arrest of development in the curtains of the valve, at an early period of intra-uterine life. He met with it three times in the aortic valves of children whose age did not much exceed four years, and in whom every other part of the organs of circulation was perfectly normal; and once saw this condition of the valve in an infant which died at, or very soon after, birth; indeed, it has been observed in more than one child of the same family.

When the apertures in the valve are very small, and few in number, they exercise no injurious influence upon the circulation and give rise to no physical sign whatever. Out of the large number met with by M. Bizot, not one presented during life symptoms of disease of the heart; this he explains by the apertures being near the free margin of the arterial valves, which naturally overlap one another. If the apertures are large, regurgitation will take place—into the ventricles, if the aortic or pulmonary valves are engaged—into the auricles, if the mitral or tricuspid valve is its seat, when a blowing or musical murmur will be developed.

*Atrophy of the Valves.*—Under the name atrophy, Dr. Kingston† has described a peculiar condition of the mitral and tricuspid valves, consisting in a shortening of the curtains in the direction of their length (the orifice preserving its normal size), without any diminution of transparency, or other alteration, except that they are somewhat thinner than natural, and a cribriform condition of the curtain of the valve may be present at the same time. Dr. Kingston met with shortening of the mitral valve in five instances; of the tricuspid in five, also; and of both on two occasions; in some of these cases the shortening was very considerable. He does not look upon it as a congenital malformation, but thinks it occurs after birth, as in ten cases the patient had reached middle age, or advanced life, before symptoms of obstructed circulation manifested themselves.

\* Guy's Hospital Reports, No. 14.

† Med. Chir. Trans. vol. xx.

*Rudimentary condition of the Valves.*—In cases of malformation of the heart, where the arterial orifices are contracted, the sigmoid and semilunar valves are often imperfectly developed, and various abnormal conditions of them have been met with. These are fully described (so far as the pulmonary artery is concerned) in the admirable series of papers by Dr. Chevers\* to which I have already alluded. “It is probable (he observes) that although a normal valvular apparatus is absent in these cases, regurgitation of blood into the ventricle occurs in but a very small proportion of instances. The arterial aspect of the contracted orifices is occasionally clothed with small, distinct masses of organized vegetations, which freely admit of the passage of blood into the artery, but afterwards close together, and prevent reflux.” The valves of the aorta or pulmonary artery are sometimes fused together, forming a funnel-like projection upwards into the vessel; in these cases, the mechanism by which regurgitation is prevented is “similar (Dr. Chevers says) to that of the small glass ink-bottles which may be carried open in the pocket without escape of the fluid;” the only difference being that the sides of the funnel-shaped valve are pliable, and capable of being pressed together.

*Irregularity in the Number of the Valves* is observed only at the arterial orifices, and is either accompanied by congenital malformation of other parts of the heart, or by contraction of the orifice; or it constitutes the sole peculiarity. The irregularity may consist in increase or diminution in the number of the valves; thus, occasionally, only two semilunar valves are present, the third being absent or rudimentary; at others, there are four semilunar valves. Increase in the number of the valves is more frequent at the pulmonary than at the aortic orifice. M. Bizot mentions a case where there were *four* pulmonary valves, two of which were much smaller than the others. Dr. Theophilus Thompson† gives a case where four pulmonary valves, all of equal size, existed; and Meckel mentions another. A preparation is preserved in the Museum of the Royal College of Surgeons in Ireland, in which there are four distinct and perfect pulmonary valves; the heart was that of a middle-aged female, and the rest of the organ perfectly normal. The same museum contains another preparation, in which there are four distinct and perfect aortic valves, and no

\* Lond. Med. Gaz.

† Med. Chir. Trans. vol. xxv.

deviation from the normal state is observed in any other part of the heart. Dr. Peacock\* met with nine examples of excessive development of the valves, in the museums of the London college of Surgeons, of St. Thomas, St. Bartholomews, Guy's Hospital, and in his own collection; in eight of these the excess was at the pulmonary orifice; in one only at the aortic orifice. The following are his conclusions from an examination of these specimens:—

1. “In some cases the excess of the number of valves seems to be due to the division of one of them into two; such divided valves being smaller in size than the others.”

2. “In other cases, there are three valves of nearly equal size, with a smaller supplementary valve interposed between two of them.”

3. “Occasionally, the aperture is provided with four valves, gradually decreasing in size; and,

4. “In the other cases, there may be four valves of nearly equal size and natural form.”

The instances in which only *two* arterial valves were present are not uncommon. Dr. Barclay's table† contains seventy-nine cases of valvular disease; in four of these but two aortic valves existed. Dr. Taylor records a case where the aortic and pulmonary orifice had each but two valves. M. Bizot and Mr. Paget each met with cases where only two pulmonary valves were present, and Dr. Graves has recorded another. It is remarkable that in Mr. Paget's case, in two of Dr. Barclay's, and in Dr. Graves', there was evidence of recent endocarditis, the malformed valves being coated with recent lymph; illustrating the correctness of the remark made by Mr. Paget,‡ that “parts which from defective or erroneous development are wrongly shaped, are often at the same time imperfect in their tissue, and therefore very liable to disease.” This subject has been investigated by Dr. Peacock§ with his usual ability. In the museums alluded to, he met with forty-one examples of defective development of the arterial valves; in nine, the malformation was at the pulmonic orifice, in thirty-two at the aortic; in one instance there were only two valves at both the aortic and pulmonary orifices. In seventeen of these cases, “the

\* Trans. Path. Soc. 1851-52.

† Med. Chir. Trans. vol. xxxi.

§ Trans. Path. Soc. 1851-52.

‡ On Obstruction of the Branches of the Pulmonary artery.

valves had subsequently become the seat of such extensive disease that their original condition could not be clearly ascertained; of the remaining twenty-four, with the exception of three or four, all afforded evidence that the orifices had originally been provided with three distinct valves."

According to Dr. Peacock, "in by far the more frequent form of malformation, there exist only two valves, and the defect in the number is apparently due to the adhesion of the contiguous sides of two of the valves, so that, in the process of development they have become blended into one." In another form, "the original triple condition of the valves is indicated by a small rudimentary valve interposed between the two others." Cases do occasionally occur (he adds) in which the aortic orifice is found to be provided with only two valves; the deficiency being, however, occasioned by the adhesion of two of the valves together from disease after birth, and the subsequent ulceration, or atrophy of the septum so formed, or by the breaking down of the angle of attachment of two of the valves."

*Congenital Arctation of the Arterial Orifices* is not very uncommon at the right side of the heart, and is very generally associated with malformation of some other part of the heart, and often with *cyanosis*, as shown by Dr. Craigie. We are principally indebted to M. Tiedemann,\* Dr. Craigie,† and Dr. Chevers, for our knowledge respecting it.

In arctation of the orifice of the *pulmonary* artery, some imperfection of the valves at the mouth of the vessel is usually also present, several varieties of which have been figured by Dr. Chevers. The state of parts most frequently met with according to him is "where the pulmonary orifice is furnished with an imperfect valvular apparatus in the form of a transverse or conical partition, having an aperture in its centre, and presenting a smooth surface below, but above three slightly elevated ridges, which are apparently rudiments of the free edges of the sigmoid valves." A very similar appearance is occasionally seen at the *aortic* orifice; "in some instances, the mouth of the artery is occupied by a thimble-shaped membrane, perforated at its apex, and smooth on both surfaces; in others, the obstruction is due to excrescences adhering to the valves, or to warty or ossific elevations." Cases

\* On Arctation and Closure of Arteries.    † Ed. Med. and Surg. Jour. vol. lx.



have also been recorded where the vessel was constricted as if by a cord immediately above the valves, or the constriction was accompanied by the absence of valves, or the orifice was furnished with only two valves.

“These morbid conditions are evidently due (Dr. Chevers observes) to lesions of the vessel occurring at a very early period of intra-uterine life; the obstruction thus produced checking the further development of the parts, and frequently preventing also the due formation of the ventricular and auricular septa, and other portions of the cardiac apparatus, which are at that period either wholly undeveloped, or but imperfectly formed.” “It is remarkable (he adds) how extreme a degree of narrowing of the pulmonary orifice may exist without producing fatal interruption to the circulation. I have never seen or heard of any instance where the area of the aortic opening had become reduced to less than the diameter of a common writing quill, but a considerably greater degree of arctation of the pulmonary orifice has been noticed even in persons above the age of ten years, and this too in cases where it was not observed that any supernumerary arteries communicated with the lungs.”

When the pulmonary orifice is congenitally contracted, the following are the malformations of other parts of the heart usually associated with it, examples of which have been given by Dr. Chevers.\* 1. “The auricular and ventricular septa imperfect; the ductus arteriosus remaining permanently open. 2. The foramen ovale and arterial duct permanently open; the ventricular septum being complete. 3. The arterial duct and ventricular septum pervious; the foramen ovale perfectly obliterated. 4. The arterial duct closed, the ventricular and auricular septa perforated. 5. The ventricular septum only, incomplete. 6. Patency of the foramen ovale.”

#### RUPTURE OF A VALVE.

Rupture of a valve, first described by Corvisart, constitutes a form of partial rupture of the heart, and the curtains of the valves, the tendinous cords, or the fleshy columns may be its seat. Like rupture of the parietes of the organ, it is most common in cases where the texture of the part had been altered by previous disease, though it has also occurred in hearts otherwise apparently sound.

\* Morbid conditions of the pulmonary artery.

In noticing the effects of inflammation we said that inflammatory softening occasionally ends in ulceration and rupture of a valve; in this place, however, we have to consider ruptures independent of inflammation.

Rupture or laceration of a healthy valve is a rare accident, and the cases on record are few. Four, in which the semilunar valve of the aorta suffered, have been recorded by Dr. Quain;\* and, in a recent memoir, Dr. Peacock† has collected all the recorded cases, amounting, with one given by himself, to eleven.

The valves in which rupture has been observed are the mitral and aortic, very seldom the tricuspid. When the mitral valve is in its seat, a fleshy column may be torn across at its base, or in some other part, or the tendinous cords may be the seat of the rupture. When the semilunar valves of the aorta are ruptured, the injury may consist in "the angle of attachment of two of the valves being torn," or "the convex margin of the valve may give way." In the cases collected by Dr. Peacock, the aortic valves were the seat of the laceration in six; the fleshy columns of the mitral valve in four; and a fleshy column of the tricuspid valve in one. The injury in all followed some considerable muscular exertion; the subjects were adults—one was a female, the rest males—and all were at the "period of life when violent muscular exertions are most frequently made."

Rupture of a valve, unlike rupture of the parietes of the heart is never instantly fatal; and the symptoms which supervene upon the accident are sufficiently characteristic. The individual is generally at the moment conscious that something has given way internally; he is attacked with acute pain, referred to the præcordia region or spine, or extending from one to the other, accompanied by paleness of the surface, oppression, dyspnoea, palpitation, sometimes syncope, and sometimes cough with hæmoptysis. Followed by signs of obstruction, or of regurgitation at the aortic orifice, if it is the seat of the rupture; or by signs of mitral regurgitation, if the latter valve has suffered rupture. "It seems (Dr. Peacock says) that when the aortic valves are injured, syncope is one of the most prominent symptoms; while in cases of suddenly-induced incompetency of the mitral valve, the patient suffers most from a sense of suffocation and oppression in the chest." Dr. Quain men-

\* Ed. Month. Jour. Dec 1846.

† Ibid, July, 1852.

tions a peculiar sound heard by the patient immediately after the accident (rupture of the aortic valves), which extended up the chest and neck, and continued to be audible for several days.

These symptoms may "subside in a short time, and be followed by those of inflammation;" but sooner or later, symptoms of valvular disease at the affected orifice set in; which usually runs a more rapid course than valvular disease from other causes; and is often attended by a great amount of distress and suffering.

#### ANEURISM OF THE VALVES.

It sometimes happens that the curtain of a valve is distended into a pouch or sac, which may be formed by all the layers which enter into the tissue of the valve, or by one layer alone after ulceration or rupture of the others. These saccular dilatations get the name of aneurism of the valves; they are met with at both the mitral and aortic orifices, and sometimes more than one exists in the curtain of the same valve. When they occur in the tricuspid valve, some congenital malformation usually accompanies them.

Aneurism of a valve, in its slightest form, consists simply in a little pouch-like dilatation of the curtain of a valve, the convexity of which in the aortic valves points towards the left ventricle, in the mitral towards the left auricle. In its more perfect form it consists of a little sac, which may be globular or flask-like in shape, or irregular; and may equal a pea or bean in size, or may attain the size of a walnut; the orifice may be smooth and circular, or jagged and irregular. They seldom contain fibrinous deposit, and they are occasionally the seat of perforations. They are not seen in valves otherwise perfectly healthy, and they may be associated with the results of inflammation, with atheromatous or calcareous deposit, or with congenital malformations of other parts of the heart. The sac may be formed by all the layers of the valve; in general, however, it is formed by a single layer, and this occasionally exhibits perforations; hence, aneurism of the valves may be accompanied either by symptoms of obstruction at the affected orifice, or by those of regurgitation through it, or by a combination of both.

## CHAPTER XIV.

## SYMPTOMS OF VALVULAR DISEASE.—PRIMARY SYMPTOMS.—SECONDARY SYMPTOMS.—TERTIARY SYMPTOMS.

VALVULAR disease presents three sufficiently well marked stages, which succeed each other pretty regularly.

The *first* includes the period antecedent to dilatation, when the disease is limited entirely to the valves.

In the *second* dilatation of one or more of the chambers of the heart, or hypertrophy of the walls is superadded to the valvular disease.

The *third* commences with the period when other organs become engaged as the result of the impediment to the pulmonary or general venous circulation, and terminates with the death of the patient.

The symptoms characterizing these several stages are different, and the terms *Primary*, *Secondary*, and *Tertiary* may be used to distinguish them.

1. *The Primary Symptoms* are the result of the modifications which the current of blood undergoes in its passage through the diseased orifices of the heart, and are furnished mainly by auscultation.

2. *The Secondary Symptoms* are superadded when dilatation of the cavities, or hypertrophy of the walls of the heart have supervened upon the valvular disease.

3. *The Tertiary Symptoms* set in when the impediment to the circulation has led to congestion of the lungs, liver, gastrointestinal mucous membrane, kidneys, or brain; or, to transudation of serum into the cellular tissue, or serous cavities.

## PRIMARY SYMPTOMS OF VALVULAR DISEASE.

These, which are mainly furnished by auscultation, are not only the first to indicate imperfection of the valvular apparatus, but they are the most precise and positive; and by their aid alone the diagnosis of valvular disease, in any of its stages, may generally

made. The effect of disease upon the valves and orifices of the heart, we have seen to be to contract, deform, or otherwise alter the orifices, or to prevent the valves from fulfilling their valvular functions; and as a consequence, the normal sounds of the heart are obscured, or masked by other and different sounds, or they are replaced by new and abnormal sounds.

The abnormal sound most frequently heard under these circumstances, and the one almost always first audible is *bruit de soufflet*; may preserve this character throughout, or it may pass into a *ring*, *sawing*, or other rough murmur, or it may be replaced by musical murmur; and it will facilitate our tracing these morbid sounds to their source, and determining their seat, if we bear in mind that a valvular murmur, audible at the period of the first sound of the heart, must be produced by the passage of the blood *out of a ventricle*; that a murmur heard at the period of the second sound must be produced by the passage of the blood *into a ventricle*; and, as valvular murmurs are in a great measure limited to the right side of the heart, and the left ventricle has but two orifices, a murmur replacing or obscuring one or other of the normal sounds will give its seat, in the majority of cases, at either the aortic or mitral orifice. As a general rule, likewise, valvular murmurs which accompany the ventricular systole, and the passage of the blood out of a ventricle are louder, harsher, or more intensely blowing than those which accompany the ventricular diastole; the latter are usually *soft* and blowing, sometimes hissing, and occasionally musical. If any difficulty is experienced in determining whether a murmur is systolic or diastolic, it should be explored from the apex upwards; the sounds of the right side of the heart ought then to be traced to the left side of the præcordial region; the finger at the same time being kept upon the carotid artery, or upon the point at which the apex of the heart is felt to beat.

Each ventricle having two orifices, theoretically each orifice might be the seat of either an obstructive or regurgitant murmur; this would make eight valvular murmurs to be distinguished from one another; in practice, however, they are almost limited to three, which, in the order of their frequency are the mitral regurgitant, the aortic regurgitant, and the aortic obstructive.

*Aortic orifice—Obstructive disease.*—When the orifice of the aorta is contracted, as the result of disease of its semilunar valves,

or of any of the morbid conditions already described, by which the outward current from the ventricle is impeded, or the surface over which the blood passes is rendered uneven or rough, the normal *first sound* of the heart will be converted into a murmur, which has a blowing or whistling character, or the rougher quality of sawing or rasping, according to the nature and amount of the obstruction, according to the shape of the diseased orifice, and particularly, according to the force with which the blood is propelled by the left ventricle.

The aortic obstructive murmur always takes the place of the normal first sound of the heart, and is synchronous with the impulse and the pulse. It is best heard opposite the semilunar valves of the aorta, and from this point upwards in the direction of the large arteries which come off from the arch; while it diminishes in intensity as we carry the stethoscope downwards to the apex of the heart. Its character varies considerably in different cases, and it may be absent altogether although a considerable amount of disease exists; thus, if the parietes of the left ventricle are attenuated or softened, the blood will be propelled with too little force to develop a murmur, or if the orifice becomes so much contracted as to admit a very small current of blood a murmur may likewise be inaudible. The intensity of this sound is influenced, in some respects also by the nature of the obstruction and its shape, thus a rigid semilunar valve projecting into the aperture, or a vegetation hanging from one of the valves will give rise to a louder murmur, *cæteris paribus*, than a simply contracted state of the aperture.

The aortic obstructive murmur might be mistaken for the murmur of mitral regurgitation, which is heard at the same period of the heart's action; the latter, however, is louder, more prolonged, and often more intensely blowing; it is, besides, best marked at a different part of the præcordial region.

*Aortic orifice—Regurgitant disease.*—When any of the abnormal conditions already mentioned, are present, by which the closure of the semilunar valves of the aorta is prevented, a murmur will be audible at the period of the ventricular diastole, and *second sound* of the heart. This murmur, as a general rule, is soft and blowing, always more prolonged than the normal second sound of the heart, and is best heard from a point opposite the semilunar valves to the apex of the organ. Occasionally it has a musical, whistling or hissing

character, very rarely that of sawing; the different degrees of intensity which it presents, depend upon the size and shape of the aperture which permits regurgitation, upon the strength of the reflux current of blood, and the size of the cavity into which regurgitation occurs.

*Aortic orifice—Obstructive and Regurgitant disease.*—The same morbid conditions of the aortic orifice or its valves, which cause an impediment to the outward current of blood from the ventricle, may, in addition, prevent the perfect closure of these valves; and the physical signs of regurgitation may be combined with those of obstruction. Under such circumstances a double murmur (*systolic* and *diastolic*) will be audible, which presents the characters either of these murmurs, when single, may have.

Although the morbid states of the aortic orifice or its valves, which permit regurgitation, are often such as must cause some impediment to the outward current of blood from the ventricle, a single is more common than a double murmur, and this is usually diastolic.

A murmur replacing the double sound of the heart, but depending upon a different cause, is by no means uncommon, and is liable to be mistaken for that last noticed. For instance, when the ascending portion of the arch of the aorta is somewhat dilated, when its lining membrane is uneven, and rough from adventitious deposit, and its coats rigid, and inelastic from the same cause, a double murmur will be audible over the arch of the aorta, accompanied by visible pulsation in the arteries of the neck and upper extremities, and by the jarring pulse of aortic regurgitation. It may be distinguished from the double murmur of aortic regurgitant disease by being loudest over the first bone of the sternum, and by its character, which is short, rough, and sawing, not prolonged or blowing.

*Mitral orifice—Obstructive disease.*—Disease of the mitral valve or orifice, causing an impediment to the current from the left auricle into the left ventricle is sufficiently common, but a diastolic murmur from this cause is excessively rare—so rare that the most excellent observers have never heard it. Indeed, the current from the auricle into the ventricle is generally too feeble to develop one, and this condition of the mitral valve and orifice is characterized rather by a diminution in the intensity of the second sound, than by a murmur; and, if the contraction is extreme, the second sound may be scarcely audible.



M. Skoda, however, lays down as the ordinary sign of this lesion, "a murmur replacing the second sound in the left ventricle, which is often so prolonged as to be only momentarily interrupted during the heart's systole." By others it is described as a soft blowing murmur, loudest towards the apex of the heart, and rather following than accompanying the second sound. I cannot help thinking that in the cases in which a murmur from this cause is said to have been heard, the murmur of mitral regurgitation was mistaken for it, which sometimes, instead of accompanying the ventricular systole and the first sound, *follows it*, and thus obscures the second sound of the heart.

*Mitral Orifice—Regurgitant disease.*—While a direct mitral murmur is one of the rarest abnormal sounds, a systolic regurgitant murmur at this orifice is one of the commonest, and will be heard when, from any cause the left auriculo-ventricular orifice is imperfectly closed during the ventricular systole. The mitral regurgitant murmur corresponds with the impulse, and the pulse; is audible at the period of the ventricular systole and *first sound* of the heart, and is generally sufficiently loud to mask the latter. As a general rule it has a blowing character, occasionally it is rough, grating, or whistling; it is usually a loud and prolonged sound, contrasting remarkably with the softer murmur of aortic regurgitation.

We occasionally meet with some remarkable varieties of this murmur; thus it may occupy not only the whole period of the first sound of the heart, but it may extend into the period of the second sound, which it will obscure, when merely a *single blowing sound* will be heard; at other times it seems rather to follow the first sound, and to occupy the interval between the two sounds, which are heard independent of it. In some cases (both of rheumatic and non-rheumatic origin), which I have watched from the commencement, the mitral regurgitant murmur was preceded for some time by a *triple* sound, which gradually passed into the murmur; the triple sound was sometimes a reduplication of the first sound, sometimes of the second, and in one instance a *quadruple* sound was heard. The triple sound eventually passed into a murmur, and afterwards occasionally alternated with it.

The mitral regurgitant murmur has usually its greatest intensity about the apex of the heart; it may be audible below this point,



ut it is not propagated upwards in the course of the large vessels ; in thin subjects, it is usually audible also in the left axilla, and in the region of the left scapula. The loudness of this murmur depends upon the force of the regurgitating current of blood, while its tone depends upon the shape and size of the aperture which permits regurgitation, and the rigidity of the curtains of the valve. The loudness or intensity of this murmur is not by any means, however, a measure of the advanced stage of disease of the valve ; a small aperture permitting regurgitation, in an otherwise healthy heart, is often accompanied by a loud bruit de soufflet, or by a musical murmur, while a very advanced stage of disease of this valve may be unattended by any murmur whatever. Mr. O'Ferrall\* first called attention to this disappearance of the murmur of mitral regurgitation ; he explains it by supposing "that a valve so shortened as to be incapable of closing the normal opening may become adequate to its task in consequence of progressive contraction, *combined with* a favourable adaptation of the aperture itself." In the cases which I have met with, however, the disappearance of the murmur was due to the gradual contraction of the orifice, which at length became incapable of permitting the passage of a reflux current, sufficient to generate a murmur.

The murmur of mitral regurgitation was first described by Dr. Elliotson† in the year 1830 ; Dr. Hope, however, says it was first recognised by him in the year 1825, "a murmur from regurgitation at any of the orifices of the heart, according to him, being then unknown."

*Pulmonary Orifice—Obstructive disease.*—A systolic murmur at the pulmonary orifice, the result of disease of its sigmoid valves is very rare ; Dr. Williams met with but three cases in which he suspected the existence of disease in these valves during life, and Dr. Ormerod‡ has recorded three others ; a systolic murmur was audible in each, and in two of the latter all the other valves of the heart were healthy. He is of opinion that a systolic murmur "not traceable along the aorta, but clearly audible in a line from the third left intercostal space at the edge of the sternum towards the middle of the left clavicle for the distance of two to two and a-half inches," will probably have its seat in the pulmonary artery.

\* Dub. Jour. of Med. vol. 23.

† Lumleyan Lectures 1830.

‡ Ed. Med. and Surg. Jour. vol. 65.

Independent of disease of the pulmonary valves, a systolic murmur is occasionally audible at this orifice owing to the calibre of the vessel being diminished by pressure from without, as in young subjects in whom the parietes of the thorax are narrow, if strong pressure with the stethoscope is made, as first noticed by Dr. Latham; or by pressure from within, as by the growth of an aneurismal or other tumor, by a morbid growth from the pericardium as noticed by Dr. Elliotson, by enlarged bronchial glands, or by a solidified portion of lung compressing the vessel, to which Dr. Latham first called attention. An inorganic murmur is also occasionally heard at this orifice, which may or may not be associated with an aortic murmur, and has generally an anæmic origin.

According to M. Skoda,\* “hypertrophy with dilatation of the right ventricle caused by defect of the mitral valve, is occasionally accompanied by a systolic murmur in the pulmonary artery,” which he attributes to “softening of the inner membrane of the distended artery;” Zehetmayer thinks it depends on “corrugation of the lining membrane of the vessel;” while Weber,† who notices the murmur, says he is unable “to contribute aught towards its explanation.”

*Pulmonary Orifice—Regurgitant disease.*—A diastolic murmur at the pulmonary orifice is extremely rare, so rare that the most experienced auscultators have never heard it. The most frequent abnormal condition of the sigmoid valves of this vessel is a cribriform condition of their curtains, which is nearly as often met with here as at the aortic orifice, and must permit of the reflux occasionally of a small quantity of blood; but a murmur from this cause is scarcely ever heard, because from the shortness of the trunk of the pulmonary artery, and the course which its divisions take, the blood does not regurgitate into the right ventricle with sufficient force to develop a murmur. Besides the apertures in the curtains of the valve are usually very small, and so situated that regurgitation seldom can take place; while a cribriform condition of these valves is almost always associated with a similar malformation of the aortic valves, when a murmur if developed, would be masked by the louder one of aortic regurgitation.

*Tricuspid orifice—Obstructive disease.*—Contraction of the

\* Translation by Dr. Markham.

† Manual translated by Dr. Cockle.

the auriculo-ventricular orifice is rare, and a diastolic murmur at this orifice is not known.

*Tricuspid orifice—Regurgitant disease.*—A dilated state of the tricuspid orifice, permitting regurgitation, is very common, and is usually the ultimate result either of valvular disease at the left side of the heart (particularly mitral contraction) or of chronic bronchitis with emphysema of the lungs; it is almost always accompanied by dilatation with hypertrophy of the right ventricle, and the phenomenon known as *jugular pulsation* is usually present in such cases. As a general rule, however, it is not attended by a systolic murmur, the current being too feeble to develop one.

The site of a tricuspid regurgitant murmur when present is at the lower end of the sternum, or a little above or below this point, and at the lower margin of the ribs close to it; it is inaudible or nearly so at the apex of the left ventricle; its character is blowing, but softer than the murmur of mitral regurgitation. Even when present it may be difficult to be detected, being masked by the louder murmur of mitral regurgitation which often accompanies it, and of which it appears to constitute a part.

In examining cases of valvular disease, it is occasionally difficult to pronounce at once whether a murmur is systolic or diastolic, particularly if the heart's action is irregular or tumultuous, or when the pulse is extremely rapid; and repeated examinations may be necessary before an exact diagnosis can be made. Such cases are, however, exceptions to the general rule, and are never examples of simple valvular disease, but are complicated with degeneration of the muscular fibre of the heart, or with disease of other parts of it, or other organs.

When the two sets of valves are diseased at the same time, and a murmur accompanies each, the diagnosis, though more difficult than when a single valve is engaged, may still be made, by attending to the rules laid down, particularly by the character of the murmurs, the situation at which each is best heard, and the direction in which each is propagated. In this way, the aortic regurgitant and the mitral regurgitant murmur can be diagnosed with comparative facility, although the signs of each are less characteristic than when they exist singly. When contraction of the aortic orifice is combined with regurgitation at the mitral orifice, the murmurs occur at the same period of the heart's action, and

the latter may from its greater intensity mask the former; still by careful and repeated examination, the diagnosis may generally be made. When contraction of the aortic orifice is combined with the same condition of the mitral orifice, the diagnosis may be one of considerable difficulty, unless we have had the opportunity of watching the case from the outset. The exact determination of the point will, however, make little difference in the treatment.

#### VISIBLE PULSATION IN THE LARGE ARTERIES.

Visible pulsation in the large arteries of the neck and upper extremities was first noticed as a sign of cardiac disease by Dr. Abercrombie;\* he has given the details of a case where “a remarkably strong but regular pulsation of the whole arterial system, particularly a peculiar and strong jarring of the carotids and subclavians” was present. On examination after death, one of the aortic valves was “perforated by an irregular opening which occupied the greater part of it, and gave an appearance as if it had been torn from its attachment along its base.” In his commentary upon it, Dr. Abercrombie observes, “the prominent symptom in this case was the peculiarly strong pulsation of the arterial system, especially of the large arteries about the neck. The pulsation of the heart did not by any means correspond with it.” Dr. Corrigan† was, however, the first to connect this sign with patency of the aortic valves. “When a patient affected with this disease is stripped, the arterial trunks of the head, neck, and superior extremities, immediately catch the eye (he observes) by their singular pulsation. At each diastole of the arteries, the subclavian, carotid, temporal, brachial, and, in some cases, even the palmar arteries are suddenly thrown from their bed, bounding up under the skin.” This phenomenon is, in general, best marked, after the carotids, in those arteries which are most superficial, as the radial, brachial, and temporal, which sometimes, in Dr. William’s words, appear “like worms under the skin, wriggling into tortuous lines at each pulse.”

Visible pulsation in the large arteries is commonly supposed to be *pathognomonic* of aortic regurgitation; this, however, is by no means the case; it is observed also in,

\* Trans. of Med. Chir. Soc. of Ed. vol. i. 1824.

† Ed. Med. and Surg. Jour. vol. xxxvii. 1832.

1. Diseased states of the lining membrane of the arch of the aorta, with dilatation of this part of the vessel.
2. In some forms of aneurism of the arch of the aorta.
3. In certain cases of anæmia, particularly in the variety accompanied by enlargement of the thyroid gland.
4. In old subjects in whom the coats of the arteries have become rigid.

In some of these states the visible pulsation is evidently, and all it is probably, the result of regurgitation into the arch of the aorta, from the large vessels given off by it. Dr. Henderson\* says he has repeatedly observed this phenomenon "in mere eccentric hypertrophy of the left ventricle;" and he adds that "it may not be present in a very considerable degree in cases of incompetency of the aortic valves." Dr. Blackiston also records cases where it is present, although the aortic valves did not permit regurgitation; he refers it, then, to hypertrophy of the heart. I do not think that visible pulsation of the large arteries is ever present in mere hypertrophy of the heart; and it is probable that in the cases referred to by Drs. Blackiston and Henderson, some one of the morbid conditions noticed above, existed in addition to the hypertrophy. Indeed, in one of Dr. Blackiston's cases, atheromatous disease of the ascending portion of the arch of the aorta was present, and a short, double saw sound (which I have shown to be characteristic of the latter lesion) was audible likewise.

#### INDICATIONS AFFORDED BY THE PULSE.

In valvular disease at the right side of the heart, the pulse affords no assistance to the diagnosis: in certain forms of valvular disease at the left side, however, it presents well-marked characters.

*Obstructive Disease at Aortic Orifice.*—The pulse here is little altered, unless the impediment to the passage of the blood is such as to diminish considerably the calibre of the orifice; because the walls of the left ventricle usually become hypertrophied, the result of this lesion, and what is wanting in quantity, is made up by the strength of the ventricular systole. When the obstruction becomes extreme, which however is rare, the pulse becomes small, and occasionally intermittent or irregular.

*Regurgitant Disease at Aortic Orifice.*—When the aortic

\* Ed. Month. Jour. May, 1843.

valves permit free regurgitation, the pulse is jerking and receding, but its rhythm is regular, and its frequency is not increased; at the same time the pulsation of the arteries of the neck and upper extremities is visible and locomotive. The thrill becomes more perceptible if the finger is applied to a larger artery than the radial, as the brachial; and if we lay two or three fingers upon the line of the artery. These characters of the pulse are not, however, limited to cases of aortic regurgitation; they are observed likewise, in some forms of aneurism of the ascending or transverse portion of the arch of the aorta, and in dilatation of this part of the vessel, with disease of its coats; while in aortic valve disease, if the aperture which permits regurgitation is very small, it may have no influence upon the pulse.

Another peculiarity in the pulse of aortic regurgitation, to which attention was first directed by Dr. Henderson,\* is, that the interval between the impulse of the heart and the radial pulse is longer than natural. "In many instances, the interval is so much prolonged, that the heart and the radial artery seem (he says) to beat with a distinct alternation." I have, in a few instances, met with this peculiarity in the pulse; Dr. Douglas,† however, looks upon it as a doubtful sign: according to him, it does not occur except in conjunction with more or less dilatation.

*Obstructive Disease at Mitral Orifice.*—When the left auriculo-ventricular orifice is contracted, the passage of the blood from the auricle into the ventricle being impeded, the pulse is smaller and weaker than natural, according to the decree of the contraction, and the amount of obstruction; if this is very considerable, the pulse, in addition, becomes intermittent, or unequal and irregular. This state is frequently combined with a condition of the valve permitting regurgitation, when the contrast between the strength of the impulse of the heart and of the pulse at the wrist is very remarkable. In addition, when the contraction of the orifice is extreme, the heart's impulse and the radial pulse are not always synchronous, as first pointed out by Mr. Adams.

*Regurgitant Disease of Mitral Orifice.*—When the mitral valve imperfectly fulfils its function, and permits a small amount of blood to regurgitate into the auricle at each ventricular systole, although a loud bruit de soufflet may be audible, the pulse pre-

\* Ed. Month. Jour. May, 1843.

† Ibid.

ves its normal strength : but if the auriculo-ventricular aperture much dilated, and allows a considerable reflux of blood (which, however, is rare), the pulse becomes small and weak, sometimes ermittent or irregular, resembling the pulse of considerable contraction of the orifice.

A small, weak, intermittent, or irregular pulse is not, however, peculiar to disease of the valves or orifices of the heart; it is these characters in the advanced stages of dilatation and softening of the left ventricle, it may also have them in any form of cardiac anæmia, accompanied by much embarrassment of the circulation.

The principal source of fallacy in the pulse, as a guide to diagnosis in valvular disease, arises from the circumstance that obstructive disease may be associated with a state of the same valve or orifice permitting regurgitation, or with obstructive or regurgitant disease of another valve; or, further complicated with morbid changes in the muscular tissue of the ventricles, with anæmic state of the system, with functional derangement of the heart, or a plethoric or gouty habit.

#### SECONDARY SYMPTOMS OF VALVULAR DISEASE.

When valvular disease has existed for a period which varies in different subjects, the parietes of the chamber or chambers behind the affected valve in the course of the circulation, become increased in thickness, in order to overcome the impediment; or the cavities themselves, yielding to the distending force from within, become increased in all their dimensions; in general, both these changes ensue; the secondary symptoms of valvular disease are, therefore, a combination of those of dilatation and hypertrophy with valvular disease.

When these secondary effects have ensued, the symptoms become more marked; indeed, until then, the subject of it may be unconscious of the existence of disease; and the majority of cases have reached this stage before the patient seeks advice. These secondary effects usually supervene slowly and gradually; occasionally they come on more quickly; and the more quickly they supervene, the more rapidly the disease usually runs its course. When valvular disease has its origin in endocarditis, in young subjects, the secondary effects are superadded quickly; on the other hand, when it supervenes upon atheromatous and calcareous



deposit on the valves, and the patient is an adult, or advanced in life, a much longer period usually elapses.

*Hypertrophy* is the most frequent secondary effect of certain forms of valvular disease, particularly of the obstructive lesions; it is not, however, to be regarded as an unfavourable complication unless excessive, or combined with a considerable amount of dilatation, or with softening of the muscular tissue of the ventricle, but rather as the means by which nature enables the heart to overcome the impediment to the circulation, and by which the injurious results, which would otherwise follow, are diminished—the effect of hypertrophy being to increase the propulsive power of the ventricles, the heart is then, notwithstanding the valvular obstruction, enabled to transmit the blood which it receives to remote parts—and the circulation is maintained.

*Dilatation*, on the other hand, is an unfavourable secondary effect of valvular disease; indeed, by itself it constitutes a real state of disease, and when combined with valvular disease it renders the latter a much more formidable affection, because the ability of the heart to maintain the circulation, diminishes in proportion as the ventricles are enfeebled by dilatation of their cavities; venous congestion sets in earlier, and the disease pursues a more rapid and more certain course to a fatal termination. Excessive dilatation once established, becomes, as Dr. M'Dowel well observes,\* a direct cause of obstruction. Though the dilated cavities contain much blood, they are able to receive or to expel but little, the heart becomes embarrassed and oppressed, especially when the circulation is hurried; it beats with increased rapidity to compensate for diminished power, and hence arise palpitations with irregularity of action, dyspnoea, hæmoptysis, œdema and congestion of the lungs, dropsy and perhaps an interruption of the functions of the brain. Thus it is, that an excessively dilated heart, by its inefficiency to unload its chambers, produces symptoms similar to those which are the result of obstructive valvular disease."

#### ALTERATIONS OF THE IMPULSE.

When valvular disease succeeds to acute endocarditis, the increased irritability of the heart set up at that period persists, and when it has arrived at its second stage, increased impulse becomes

\* Dublin Quarterly Journal, vol. xiv.



a permanent symptom, and according as hypertrophy or dilatation predominate, it will have the characters which appertain to either. The site of the impulse, in such cases, is more or less altered; it is felt lower down than natural, and more to the left or right side according to circumstances; and this alteration in its site may itself become a guide to the seat of the valvular lesion. Thus, when regurgitant disease of the aortic valves has existed for some time, the apex of the heart will be seen and felt to beat upon a lower plane, and more to the left side than natural (sometimes even on a line with the axilla), because the effect of the long continuance of this form of disease, is to occasion dilatation with hypertrophy of the left ventricle, by which the long diameter of the heart is increased. Again, the effect of obstructive disease at the *mitral* orifice is eventually to cause hypertrophy with dilatation of the *right* ventricle, by which the transverse diameter of the heart is increased, when the impulse will be felt more to the right side, and lower down than natural. In cases of free mitral regurgitation an undulatory impulse is sometimes perceptible from the second intercostal space downwards, if the patient's chest is narrow or contracted, and the subject is under puberty.

The strength of the impulse is ordinarily in the ratio of the thickness of the parietes of the ventricles, and the vigour of the circulation; this does not, however, hold good always in valvular disease, the impulse may be inconsiderable, irregular, or scarcely to be felt, although the ventricles are much hypertrophied, if the circulation is embarrassed, if the heart is softened, or if emphysema of both lungs is present.

When hypertrophy with dilatation has followed valvular disease, the size of the heart is necessarily increased, and a larger portion of its surface comes in contact with the parietes of the chest; the præcordial region then yields a dull sound on percussion over an abnormal extent of surface, proportionate in general to the increase in size of the organ, and varying in its site according to the part of the heart engaged.

#### TURGESCENT AND PULSATION OF THE JUGULAR VEINS.

The former of these phenomena is the most common, and may be present whenever an impediment exists to the entrance of the venous blood into the right auricle, or to the free passage of the

deposit on the valves, and the patient is an adult, or advanced in life, a much longer period usually elapses.

*Hypertrophy* is the most frequent secondary effect of certain forms of valvular disease, particularly of the obstructive lesions; it is not, however, to be regarded as an unfavourable complication unless excessive, or combined with a considerable amount of dilatation, or with softening of the muscular tissue of the ventricles, but rather as the means by which nature enables the heart to overcome the impediment to the circulation, and by which the injurious results, which would otherwise follow, are diminished—the effect of hypertrophy being to increase the propulsive power of the ventricles, the heart is then, notwithstanding the valvular obstruction, enabled to transmit the blood which it receives to remote parts—and the circulation is maintained.

*Dilatation*, on the other hand, is an unfavourable secondary effect of valvular disease; indeed, by itself it constitutes a real state of disease, and when combined with valvular disease it renders the latter a much more formidable affection, because the ability of the heart to maintain the circulation, diminishes in proportion as the ventricles are enfeebled by dilatation of their cavities; venous congestion sets in earlier, and the disease pursues a more rapid and more certain course to a fatal termination. Excessive dilatation once established, becomes, as Dr. M'Dowel well observes,\* a direct cause of obstruction. Though the dilated cavities contain much blood, they are able to receive or to expel but little, the heart becomes embarrassed and oppressed, especially when the circulation is hurried; it beats with increased rapidity to compensate for diminished power, and hence arise palpitations with irregularity of action, dyspnoea, hæmoptysis, oedema and congestion of the lungs, dropsy and perhaps an interruption of the functions of the brain. Thus it is, that an excessively dilated heart, by its inefficiency to unload its chambers, produces symptoms similar to those which are the result of obstructive valvular disease."

#### ALTERATIONS OF THE IMPULSE.

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\* Dublin Quarterly Journal, vol. xiv.

permanent symptom, and according as hypertrophy or dilatation predominate, it will have the characters which appertain to either. The site of the impulse, in such cases, is more or less altered; it is felt lower down than natural, and more to the left or right side according to circumstances; and this alteration in its site may itself become a guide to the seat of the valvular lesion. Thus, when regurgitant disease of the aortic valves has existed for some time, the apex of the heart will be seen and felt to beat upon a lower plane, and more to the left side than natural (sometimes even on a line with the axilla), because the effect of the long continuance of this form of disease, is to occasion dilatation with hypertrophy of the left ventricle, by which the long diameter of the heart is increased. Again, the effect of obstructive disease at the mitral orifice is eventually to cause hypertrophy with dilatation of the right ventricle, by which the transverse diameter of the heart is increased, when the impulse will be felt more to the right side, and lower down than natural. In cases of free mitral regurgitation an undulatory impulse is sometimes perceptible from the second intercostal space downwards, if the patient's chest is narrow or contracted, and the subject is under puberty.

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#### TURGESCENT AND PULSATION OF THE JUGULAR VEINS.

The former of these phenomena is the most common, and may be present whenever an impediment exists to the entrance of the venous blood into the right auricle, or to the free passage of the

blood through or out of the right chambers of the heart. Its cause is simply mechanical, and the impediment to the return of the venous blood, from the head and neck, may be seated either at the right side of the heart, in the lungs, at the left side of the heart, or in more distant parts; and, may depend upon dilatation of the right chambers of the heart; upon obstructive disease at the mitral orifice; upon disease of the lungs or bronchial tubes, impeding the pulmonary circulation; or, upon pressure on the superior cava by an aneurismal or other tumor. Pulsation in the jugular veins will be present in addition, when the tricuspid valve permits free regurgitation, and the right ventricle is at the same time hypertrophied and dilated; a portion of the contents of the right ventricle being propelled backwards into the auricle at each systole, which reacts upon the blood contained in the auricle, and through it upon the current descending by the superior cava and its branches.

Pulsation is in general observed only in the internal jugular veins, these vessels not being provided with valves like the external; whereas distension engages both the internal and external jugulars. The pulsation is usually single, and immediately follows the ventricular systole; in some cases it is double, but the second impulse is very feeble, merely an undulation of the surface. The pulsation, though usually evident only to the eye, is occasionally sufficiently strong to be felt by the finger, several examples of which have come under my notice. It is always most marked immediately above the clavicles, and may extend some distance up the neck, if the latter is short; I have observed it to reach the angle of the jaw, only when the external jugular was the seat of pulsation. It is often perceptible upon both sides of the neck, though usually better marked on the right; occasionally it is limited to the left side.

The absence of jugular pulsation by no means proves the absence of regurgitation; in several cases of decided regurgitation, given by Dr. Blackiston,\* in which the heart was attenuated or softened, it was absent. "If both ventricles are hypertrophied, a strong venous current will (he observes) be met by an equally strong regurgitating current, the shock will be great, and the pulsations of the veins of the neck will be strongly marked. On the other hand, if both ventricles are softened or attenuated, a feeble

\* On Diseases of the Chest.

venous current will be met by an equally feeble regurgitating current, and the thrill and pulsation will be slight, whilst the flow of venous blood will be as much impeded in the one case as in the other."

#### FREMISSEMENT CATAIRE, OR PURRING TREMOR.

The valvular lesion with which this sign is most frequently associated is a state of the mitral valve and orifice permitting regurgitation; it is also, but rarely, met with in obstructive or regurgitant disease of the aortic orifice. It requires for its development a pretty strong current of blood, ceasing when the heart's action becomes enfeebled, embarrassed, or impeded, towards the close of the disease.

Whenever fremissement is felt, a murmur of some kind will be audible on auscultation; which is generally blowing, sometimes musical, or rough, or harsh; and, if the murmur ceases to be audible, as sometimes occurs in the progress of regurgitant mitral disease, the fremissement will no longer be felt. This phenomenon is, in general, evident to the hand laid lightly upon the part; and it is best marked when the heart acts vigorously; hence if in doubt respecting it, the patient (provided he can do so without inconvenience) should be made to walk up and down the room, or up and down stairs, when it generally becomes sufficiently evident.

The ordinary site of fremissement is about the apex of the heart; in thin subjects it is felt, likewise, between the cartilages of the ribs on the left side, near the sternum. It almost always accompanies the ventricular systole; and, when well marked and constant, it is a valuable diagnostic sign of organic disease. It is not, however, limited to cases of valvular disease; it is observed, also, in congenital malformations of the heart, in aneurism of the large arteries, more particularly varicose aneurism; and in dilatation of the arch of the aorta, when combined with an attenuated state of the blood. Under such circumstances, however, the site of the fremissement is different.

#### TERTIARY SYMPTOMS OF VALVULAR DISEASE.

The tertiary symptoms of valvular disease are not all limited to lesions of the valves; several of them accompany other forms of

disease of the heart, as well as various pulmonary affections; and they are preceded always by dilatation or hypertrophy of the ventricles, or by a combination of the two.

*Congestion of the Lungs.*—The lungs, from their physiological position, between the right and left chambers of the heart, must suffer when an impediment exists to the return of the blood by the pulmonary veins to the left side of the organ; the pulmonary tissue and bronchial mucous membrane become congested, and the over-distended capillaries relieve themselves by the transudation of the watery, saline, and albuminous, sometimes of the fibrinous constituents of the blood, or of blood itself, upon the surface of the bronchial mucous membrane, or into the air-cells of the lungs; or rupture of vessels may take place, with extravasation of blood upon the mucous surface, or into the pulmonary tissue.

When the air-cells, the minute ramifications of the bronchi, and the interlobular cellular tissue are infiltrated with serous fluid, it constitutes the state with which we are familiar as *œdema of the lungs*; in which, as in œdema of the cellular tissue, the serum gravitates to the most dependent parts, occupying the base and back of the lungs, and characterized by dulness on percussion, and a moist sub-crepitant râle on auscultation. It is always an unfavourable symptom in valvular disease, and if the patient did not suffer previously from dyspnoea or cough, he is sure to do so now, or if they had been present they are certain to be aggravated. When much of the pulmonary tissue is engaged, and the air-cells of the part scarcely admit of the entrance of air, the blood will be imperfectly oxygenated, and a mixture of venous and arterial blood will circulate through the system. Œdema of the lungs is not, however, limited to diseased states of the heart; it accompanies some forms of general dropsy, it is observed in chronic bronchitis and pertussis, it follows the resolution of pneumonia, and it occurs as a sequela of measles and scarlatina.

The form of valvular disease in which the greatest amount of pulmonary congestion ensues, is contraction of the left auriculo-ventricular orifice, combined with dilatation and hypertrophy of the right ventricle: here the blood is transmitted to the lungs with considerable force, while the pulmonary veins have great difficulty in emptying themselves. Under such circumstances, sanguineous engorgement of the pulmonary tissue is certain to

ensue, followed, sooner or later, by transudation of the blood itself upon the surface of the bronchial mucous membrane, or by rupture of minute vessels upon the mucous surface, or into the pulmonary tissue, giving rise to hæmoptysis, or pulmonary apoplexy. In young subjects epistaxis may take the place of the latter. In other cases serum is effused into the pleural cavities or pericardium, constituting hydro-thorax or hydro-pericardium.

In the advanced stage of valvular disease, accompanied by much pulmonary congestion, dyspnœa and orthopnœa are often more urgent symptoms than in diseased states of the lungs themselves; the breathing is extremely laborious, and the distress constant, though it still occurs in paroxysms of increased severity. The number of the respirations is not, however, in general as much increased as in cases of pulmonary disease.

*Cough.*—When congestion of the bronchial mucous membrane ensues, cough, if not present before, usually sets in; the congested state of the bronchial mucous membrane and of the pulmonary capillaries is relieved, partly by increased mucous secretion, and partly by the transudation of the serous portion of the blood into the air cells, and minute ramifications of the bronchial tubes; this can only be removed by coughing, which is thus excited and kept up; and, thus, the efforts of nature to relieve the pulmonary congestion become the source of increased distress to the patient, and, in the advanced stage of valvular disease, may constitute a serious complication; for, if the powers of life are low and the fluid accumulates, the patient may be incapable of expectorating it, and die asphyxiated.

*Pain, Angina.*—Severe pain is not an ordinary symptom of valvular disease, and when complained of is not referred to the site of the valve engaged, nor does its immediate cause lie in the morbid changes going on in the valve, but in the secondary lesions which sooner or later follow, or in the impediment to the circulation through the heart or lungs which results.

The form of valvular disease, in which pain is the most prominent symptom, is *patency of the aortic valves*, here it sometimes occurs in paroxysms of extreme severity, it shoots from the sternum to the left scapula and shoulder, extending to the arm or forearm, more rarely to the lower extremity upon one or both sides, has many of the characters of angina pectoris, or it resembles



the pain complained of in some cases of aortal aneurism. It is not necessarily accompanied by any feeling of dyspnoea, it remits or intermits, and is liable to be induced by trifling causes, such as merely passing from a warm to a colder atmosphere; walking quickly, sometimes even at a moderate pace; by mental emotion, by indigestible food, or flatulent distension of the stomach or colon. In some instances the patient referred the increase of pain to a change in the weather, as to rain; this I have also noticed in individuals labouring under aneurism of the aorta. Sometimes again the pain seems to be relieved by exercise, and it comes on when the patient is at perfect rest, or it supervenes during sleep.

In *Mitral-valve disease* pain is seldom present unless considerable contraction of the orifice exists, and pulmonary and general venous congestion have been induced; it may then become a prominent symptom but its characters are quite distinct from those of aortic regurgitant disease; the patient complains of a distressing sense of constriction, heat, or oppression in the chest, referred either to the præcordial, sternal or epigastric regions, or to all three, accompanied occasionally by a sensation of impending suffocation, the heart appearing to be struggling to get rid of the load which oppresses it. It is more or less paroxysmal, increases at irregular intervals, often at night, is always associated with the feeling of dyspnoea, and sometimes with a tendency to syncopal attacks.

In the advanced stage of mitral valve disease, where dyspnoea is a prominent symptom, the patient often complains more of want of sleep than of pain. In many such cases, the patient for days, nay sometimes for weeks, does not obtain more than a few minutes sleep, at a time, during the twenty-four hours, and this only in the most constrained posture, as sitting upright in bed, or leaning forward upon a pillow or table, or with some substance pressed against the epigastric or præcordial region. On the whole, however, pain is not a diagnostic sign of much value in valvular disease unless severe, and the patient a male, and one who would not be likely to magnify his sufferings; or, unless accompanied by the physical and other signs of patency of the aortic valves, or of contraction of the mitral orifice. When the pain is referred to the left side of the chest, and the patient is a female, it is more commonly connected with anæmia, or functional derangement of the heart than with valvular disease.



## CONGESTION OF THE LIVER.

When the venous blood cannot pass freely through the right cavities of the heart, the return of that ascending by the inferior vena cava is impeded, the capillaries of the *venæ cavæ hepaticæ* become distended, next those of the *vena portæ*; and if the cause continues in operation, the minute ducts which ultimately form the excretory duct of the liver suffer from the compression, the secretion of bile is impeded, or its escape prevented; biliary is added to sanguinous congestion, and a section of the organ exhibits the appearance which is familiar to us as *nutmeg liver*.

Next to the lungs, the liver suffers most frequently from congestion in valvular disease accompanied by much obstruction to the venous circulation; and the forms of valvular disease with which it is generally associated are contraction of the mitral orifice, and a state of the tricuspid orifice permitting free regurgitation (in the former, pulmonary always precedes hepatic congestion); it is not, however, limited to valvular disease, but is frequently present also in dilatation of the ventricles.

In congestive states of the liver the organ is soft, lacerable, and full of blood; its bulk is likewise increased, though it preserves its normal shape; the increase in size is sometimes very remarkable, the organ reaching to or below the umbilicus, and forming a swelling evident to the eye. This may occur within a comparatively short period, and within a period equally short under treatment, or independent of it, the organ may resume its normal dimensions. It may, however, remain permanently enlarged or ulterior changes may take place in it.

When the liver is congested, the patient suffers from a sense of weight in the right hypochondrium, the part is also tender on pressure; and, if the enlargement is considerable, the descent of the diaphragm being impeded, dyspnoea is increased. When biliary is superadded to sanguineous congestion, we have in addition, nausea or vomiting, loss of appetite, thirst, and colicky pains; the bowels are constipated or too lax, the urine is scanty, high-coloured, and when cool throws down an abundant deposit of lithates, and jaundice often in a slight, seldom in a marked degree, follows.

The following table, given by Dr. Williams,\* shows the progressive effects of organic disease of the heart upon the liver:

LIVER.	CHIEF SIGNS.	COMMON RESULTS.
Congestion . . .	Soft and varying enlargment	Increased secretion or deposits.
Deranged and interrupted secretion .	Bilious attacks, Jaundice, &c.	Choloemia, mal-nutrition, dyspepsia, &c.
Intestinal deposits .	Enlargement.	
— Soft, plastic .	Soft, uniform . . .	Functional disorders, general weakness, &c.
— Fatty . . .	Do. . . . .	Cachexia, dyspnœa, general dropsy.
— Hard, granular .	Hard, irregular . . .	Jaundice, cachexia, mal-nutrition, purpura.
— Contractile .	Size progressively decreases with rounded induration .	Intestinal hæmorrhages, ascites, cachexia.

*Congestion of the gastro-intestinal mucous membrane.*—The long continuance of congestion of the liver and portal system is, sooner or later, followed by congestion of the other abdominal viscera. When the gastro-intestinal mucous membrane is in a state of permanent congestion, its functions are imperfectly performed, there is a deficient secretion of gastric juice, and the digestion is consequently slow, there is loss of appetite, and usually constipation; the patient suffers, in addition, from epigastric pain, flatulent distension (the latter sometimes constituting his chief source of discomfort,) and hæmorrhoids. The congestion is, occasionally relieved, in part, by the transudation of blood upon the mucous surface, giving rise to hæmatemesis or intestinal hæmorrhage, more frequently by the escape of blood from the hæmorrhoidal vessels.

*Congestion of the Kidneys*—The effects of impeded venous circulation become evident sooner or later upon the kidneys; but this is always consecutive to hyperœmia of the other abdominal viscera. When congestion of the kidneys ensues, these organs acquire an increase in size, their colour becomes darker, their tissue more dense, and their function is more or less deranged; evidenced by lumbar pains, diminished urinary secretion, which is high-coloured, increased in specific gravity, loaded with lithates,

\* Lond. Jour. of Med., vol. ii.

and temporarily or permanently albuminous. Anasarca if not present before sets in, renal is superadded to cardiac dropsy, and the disease runs its course more rapidly.

The following *table* given by Dr. Williams, exhibits the progressive effects of organic disease of the heart upon the kidneys.

KIDNEYS.	CHIEF SIGNS.	COMMON RESULTS.
determination of blood, congestion.	Secretion, first increasing then diminished and albuminous, or bloody. Lumbar pains and tenderness.	Disordered secretion, temporary albuminuria, uræmia, &c.
increase and casting of epithelial cells, tubuli.	Soft enlargement, urine diminished and containing casts of cells, and albumen.	The same in a more confirmed degree.
gradual filling of tubuli with common granular cells, sometimes degenerating into fat.	Secretion less urinous, more albuminous.	} Permanent albuminuria, uræmia and its consequences, cachexia, spæmiæ, dropsy, &c.
induration on vessels causing partial contraction and atrophy.	Secretion less albuminous, more watery.	

*Congestion of the Heart.*—When pulmonary and general venous congestion have supervened upon valvular disease, the coronary circulation can scarcely fail to feel its effects, the return of the blood by the coronary veins is impeded, or retarded, or a mixture of venous and arterial blood is conveyed by the coronary arteries to the tissue of the heart. This cannot continue for any length of time, without affecting injuriously the function and nutrition of this organ, impairing its vigour and rendering it less capable of maintaining the circulation.

*Obstruction of the orifice of a coronary artery.*—Independent of this effect of congestion, the tissue of the heart may suffer in its nutrition in another way. Thus, in the advanced stage of disease of the semilunar valves of the aorta, the orifice of one of the coronary arteries may be encroached upon and obstructed by the extension of the morbid deposit; and the supply of arterial blood to the muscular tissue of the heart be thus seriously diminished, leading to softening or degeneration of the heart's tissue. This point will be again alluded to when I come to speak of softening and fatty degeneration of the heart.

*Congestion of the Brain.* — When the return of the venous blood to the right side of the heart from the brain and its membranes, is impeded or retarded, distension of the venous system within the cranium ensues, the arteries of the brain cannot then, as Portal long since remarked, “empty their contents into the corresponding veins, and thus will arise congestion of the brain;” indicated by headache, vertigo, noises in the ears, ocular spectra, disturbed sleep or drowsiness; which if not relieved may go on to coma, and death by apoplexy. This is not, however, as frequent an effect of valvular disease as might at first be supposed, because general venous congestion is usually relieved by the escape of the serum of the blood into the general cellular tissue; and because in the form of valvular disease in which the greatest amount of venous congestion ensues, viz: considerable contraction of the mitral orifice, a less amount of blood is transmitted to the brain at each ventricular systole than in the normal state; and thus, though the return of the blood from the brain is retarded, the organ really receives a less quantity. Besides temporary over-distension of the veins within the cranium is occasionally relieved by the spontaneous occurrence of epistaxis, particularly in young subjects. Dr. Barlow\* has recorded several cases where profuse epistaxis occurred in connexion with cardiac disease; and he thinks that this form of hæmorrhage may “often be considered as strictly pathogonomic of an obstructed circulation through the heart, as hæmoptysis is symptomatic of tuberculated lungs, or intestinal hæmorrhage of an indurated liver.”

The most frequent cause of temporary disturbance of the functions of the brain in cases of valvular disease, appears to be the transmission of imperfectly oxygenized blood to this organ. Thus in considerable contraction of, or in very free regurgitation at the mitral orifice, in which pulmonary congestion is a prominent symptom, the venous blood does not undergo the necessary changes in the lungs, imperfectly arterialized blood is carried by the pulmonary veins to the left side of the heart, and thence transmitted to the brain, causing more or less disturbance of its functions; indicated sometimes by headache, stupor, or drowsiness, so that the patient drops off asleep while we are speaking to him; at others, by transitory delirium, sometimes of a pleasing kind; the patient

\* On Disorders of the Cerebral Circulation.

occasionally fancies that things have a different hue from the natural one, they appear yellow to him; or if he wants to get out of bed, he insists on getting out at the foot, or he is with much difficulty kept in bed.

*Diminished supply of blood to the Brain.*—The cerebral circulation may, however, be disturbed in another way; for instance, if the mitral or aortic orifice is much contracted, a less amount of blood will be transmitted by the left ventricle at each systole than in a healthy condition of the parts, and the supply which the brain receives may be insufficient for its due nutrition. Dr. Law\* was the first to call attention to this point; and he has recorded cases which show that softening of the brain with hemiplegia, may occur in connexion with valvular disease, whose effect is, either directly or indirectly, so diminish the amount of arterial blood transmitted to the brain. The forms of valvular disease in which it is most likely to occur, are contraction of the mitral orifice, and next, disease of the semilunar valves of the aorta; it is rare in simple mitral regurgitation. I have never met with it myself except in considerable contraction of the mitral orifice.

In certain forms of valvular disease, the supply of blood to a portion of the brain may be suddenly diminished or cut off, followed by atrophy from imperfect nutrition, and ending in ramollissement and paralysis. For instance, we have seen that one of the results of endocarditis, is the deposition of lymph in the form of large vegetations upon the aortic and mitral valves, particularly the former; now if these vegetations have a narrow pedicle, or are loosely adherent, one of them may be detached from the valve; or if they are firmly adherent, the fibrin of the blood in its passage through the diseased orifice may be deposited upon them, and afterwards washed away; in either case the foreign body will be carried in the course of the arterial current until arrested in some vessel, the calibre of which is insufficient to admit its passage; and according to the situation of the obstructed artery, and the importance of the parts supplied by it, a different train of symptoms will result.

This subject has been investigated with much success by Dr. Sirkes,† and the cases which he has recorded, prove that ramol-

\* Dub. Jour. of Med. vol. xvii., 1840.

† Med. Chir. Trans. vol. xxxv.

lissement of a portion of the brain, may result directly from the obstruction of a cerebral artery, owing to the lodgment of a plug of fibrin within its canal. The artery obstructed in his cases was the *middle cerebral*, which he has shown to be "the most likely to arrest a portion of fibrin floating in the blood, transmitted to the brain by the internal carotid artery." "Once arrested at the angle, or within the canal of the middle cerebral artery, a mass of fibrin, if large enough to block up the vessel, becomes (Dr. Kirkes observes) at once the cause of loss of function, and subsequent atrophy to almost all that portion of the brain supplied by the obstructed vessel; for although by the arrangement of the vessels composing the circle of Willis, ample provision is made against obstruction ensuing in any of the main arterial channels of either side, *previous* to their arrival at the circle, there is comparatively little provision for an obstruction ensuing in any of the main branches, into which this arterial circle breaks up. This remark applies especially to the *middle cerebral artery*, which, if plugged up at its origin, becomes at once almost useless as a blood-vessel; for nearly all its divisions, especially those for the central parts of the brain, proceed to their several destinations without receiving any anastomosing branch, from the other divisions of the circle of Willis." . . . "The anterior cerebral artery is, by means of the anterior communicating branch, in a great measure guarded against the occurrence of a similar evil; and in this way may be explained the infrequency of softening of the anterior cerebral lobes compared with the more frequent occurrence of this condition in the parts supplied by the middle cerebral artery."

The researches of pathologists have fully confirmed the correctness of Dr. Kirkes' observations; and several cases have been since recorded, where hemiplegia has suddenly supervened, in consequence of the supply of blood to a portion of the brain being cut off by the obstruction of the artery from which it derived its supply; while in other cases the arrest of the foreign body in the main artery of a limb has been followed by gangrene, or by symptoms simulating aneurism; a very remarkable case of the latter has been recently recorded by Mr. Tufnell.\*

\* Dub. Quart. Jour. May, 1853.

## COUNTENANCE—POSTURE.

In the advanced stage of certain forms of valvular disease, the countenance is occasionally very characteristic; for instance, when obstructive disease at the mitral orifice has been followed by tricuspid regurgitation, and pulmonary, and general venous congestion, the face becomes bloated, and of a dusky hue, the lips and cheeks purple, the eyelids puffed, the eyes sometimes staring, the conjunctiva suffused, occasionally with the yellow tinge of jaundice, the lower extremities œdematous and livid, sometimes colder than natural, sometimes disagreeably warm, and the nails blue. The respiration is laborious, and the air-passages being loaded with mucus the breathing is wheezing, the jugular veins are distended, or pulsatile, and the patient is usually quite incapable of resting in the horizontal posture. On the other hand, in the next most frequent form of valvular disease, viz: patency of the aortic valves, there is no lividity or dusky hue of the lips or cheeks; the face is usually pale, and the expression that of anxiety or pain; dyspnœa is less urgent, anasarca is slower in making its appearance, it is also always a less prominent and distressing symptom.

*Posture.*—The posture which the subject of valvular disease assumes depends upon the amount of dyspnœa present, upon the presence or absence of pain, and in some respects also upon the age of the patient. For instance in obstructive disease at the mitral orifice, with tricuspid regurgitation, and dropsy of the cellular tissue or serous cavities, the patient is often quite incapable of assuming the recumbent posture, he is obliged to be propped up in bed by pillows, or he cannot remain in bed, but sits constantly in a chair with the legs upon a stool, the dyspnœa being aggravated simply by bringing the anasarca lower extremities to a level with the body. Young subjects are particularly restless and uneasy, constantly changing their position, seldom remaining for any time in bed even at night, preferring to sit in a chair, leaning forward with the head upon a table, in which position only they obtain a little sleep.

In the advanced stage of regurgitant disease of the aortic valves, when pain is a prominent symptom, and this occurs in paroxysms of extreme severity, the posture assumed will be that



in which experience has shown the patient the greatest amount of ease is to be obtained. Thus, in one case under my care, most relief was experienced from sitting upon the floor with the back pressed firmly against a wall. In another patient, who was a long time under observation, relief during a paroxysm was obtained by pressing something firmly against the lower edge of the sternum, and the patient constantly carried a stick with a broad handle, upon which he was in the habit of leaning when the pain set in. If more severe, he leaned over the back of a chair and pressed the sternum with all his strength against it; and when in bed, he lay upon his face, with some hard substance pressed against this part.

#### CARDIAC DROPSY.

When general venous congestion has arrived at its extreme limits, the systemic capillaries relieve themselves by allowing the serous portion of the blood to transude, the subcutaneous cellular tissue becomes infiltrated, and dropsy ensues, slight at first and only observed as œdema about the ankles, or in the eyelids, which increases towards evening and disappears after resting in bed, but gradually increases, extends up the limbs, or engages the face and trunk, is marked in those parts in which the cellular tissue is lax, as the scrotum or labiæ; and predominates in the most dependent parts, as the back of the trunk when the patient lies supine, or one arm, or one side of the face, when he lies upon the side. When the lower extremities become very much swollen and the cells of the cellular tissue are completely filled and distended, serum is generally effused in addition into the peritoneal cavity; and, some time before death, effusion usually takes place into one or both pleura, by which the fatal result is generally hastened.

The anasarca which attends the advanced stage of valvular disease affords the best example of the obstructive form of dropsy. The obstruction here, as we have seen, usually commences at the left side of the heart, is followed by congestion of the lungs, and this again by dilatation of the right chambers of the heart, and tricuspid regurgitation. Valvular disease may however exist for a long time without dropsy supervening if pulmonary and general venous congestion do not ensue, or, if anœmia, debility, or renal disease are not superadded; but if the impediment to the circulation is sufficient to produce congestion, dropsy in general is not long in following.



The form of valvular disease in which dropsy ensues earliest and in which it eventually becomes the most prominent symptom, is contraction of the mitral orifice, as this lesion occasions greater impediment to the *arrière* circulation than any other. It is, as a general rule, preceded by dilatation of the right chambers of the heart and tricuspid regurgitation; indeed Dr. Blakiston\* looks upon regurgitation at the tricuspid orifice as the essential cause of the general obstruction which leads to dropsy; "tricuspid regurgitation almost invariably precedes (he says) the appearance of dropsy, whether the valves on the left side of the heart are diseased or not." Dropsy does not supervene so early, and when it does occur, it is scarcely ever so prominent a symptom in the other forms of valvular disease; indeed cases of aortic regurgitant disease frequently terminate fatally, either without the occurrence of dropsy, or with merely œdema about the ankles or face.

The period which intervenes in valvular disease before the occurrence of dropsy is very variable, and depends upon a variety of circumstances, as the nature, extent, and seat of the valvular lesion, its cause, and whether it has been followed by dilatation or not; the condition of the patient's health, his habits whether intemperate or otherwise, his occupation, station in life, &c. As a general rule, the earlier that venous congestion ensues, the sooner will dropsy make its appearance. In most instances the dropsy is at first amenable to treatment, eventually, however, remedies fail to produce their specific effects; renal is superadded to cardiac dropsy, and the patient sinks; the fatal result being often accelerated by pneumonic or bronchitic attacks, or preceded by effusion into the pleural cavities.

When dropsy supervenes in valvular disease, it always, as has been said, shows itself first as *anasarca*, and it may appear, and disappear several times before it becomes permanent; or, it may continue to progress until it invades the entire subcutaneous cellular tissue. It sometimes is seen in a single lower extremity, usually the left, and may remain confined to it for a time; more generally, both lower extremities become œdematous at the same period. Sometimes, after having been a comparatively trifling symptom for a long time, the dropsy suddenly increases, and the lower extremities attain a large size within a short period, the

\* On Diseases of the Chest.

abdomen next becomes distended, and effusion into the pleura follows. In other cases, after having been perhaps several times removed by treatment, a period arrives when remedies fail to act, the anasarca gradually increases, the lower extremities and the scrotum in the male sometimes attain an enormous size; the integuments below the knees acquire a dusky congested hue, are hot to the hand, while the feet are cold, and the patient suffers much pain from the distension. Under such circumstances, irregular patches of erythematous redness often make their appearance upon some part of the surface below the knees; or erysipelas ensues, running into gangrene, and the latter will be almost certain to supervene if punctures are made at this period in the instep or leg, to give exit to the serum. Sometimes an eruption resembling *purpura*, but of a different shade of colour, appears upon the anasarcaous legs, or *vesicles* form upon them, which give way and permit of the escape of fluid; or large *bullæ* are developed upon the instep, which break and discharge a considerable quantity of fluid, with temporary relief; or the skin of the legs or thighs cracks from the over-distension, with the same result. In such cases, the strength requires to be supported; the limbs should be wrapped in warm linen, and changed as it becomes saturated with fluid. When the scrotum or labiæ are very much swollen, excoriation of these parts is liable to ensue, hastened by the contact of urine, which soon passes into ulceration, or superficial gangrene, aggravating considerably the patient's sufferings, and hastening the inevitable termination.

## CHAPTER XV.

ES OF VALVULAR DISEASE, EFFECTS OF.—PROGNOSIS IN VALVULAR DISEASE.—TREATMENT OF VALVULAR DISEASE.—TREATMENT OF THE AUTOPSY.

*Causes of valvular disease.*—In describing the anatomical characters of valvular disease, we found that its causes were very various; that it might be the result of endocarditis, of adventitious deposit, or of the changes which these deposits subsequently undergo; that it might arise from injuries, or be a congenital malformation, and finally, that it might result from more than one of these causes combined.

Among the predisposing causes, habitual intemperance upon one hand, and violent or prolonged muscular exertions, disproportionate to the strength, on the other, are by no means common. In other instances the disease may be traced to prolonged exposure to cold, or to injuries of the chest; here endocarditis is probably the immediate cause. I have had cases under my care, where prolonged immersion was quickly followed by the development of valvular disease, or where it could be directly traced to a blow upon the side, or to a fall. Renal disease is regarded by some as a predisposing cause, but it is more commonly perhaps an effect than a cause. Advanced age would appear itself to be a predisposing cause; we seldom, in examining the bodies of aged subjects, find the endocardium covering the valves in a perfectly normal condition; it is opaque, or its elasticity is diminished, or fibromatous or calcareous patches are seen upon it.

Why the valves and orifices should be the parts to suffer, rather than the other portions of the interior of the heart, appears to be due partly to the functions the valves have to perform which exposes them more frequently to injury, and partly perhaps also to the nature of the tissues which enter into their formation. "It would seem," Dr. Elliotson\* says, "to be a general law which applies

\* Lumleyan Lectures.

to all cavities and all canals, that the membrane which invests the boundaries or outlets, is more predisposed to take on disease than any other portion of their interior; thus, stricture and scirrhus of the alimentary canal affect chiefly the cardiac and pyloric orifices of the stomach, and the rectum; the lips are frequently affected with cancer; ulceration of the air-tubes is most frequent originally in the larynx and air-cells; and the neck and mouth of the uterus and bladder are commonly the original seat of the organic diseases of those organs; so inflammatory thickening and all other diseases of the lining membrane of the heart, far most frequently occupy that portion which invests the openings, and assists to form the valves."

This would not, however, explain the greater frequency of valvular disease at the left than at the right side of the heart, and various hypotheses have been advanced to account for it. Thus it has been referred:

1. To the left side of the heart circulating arterial, and the right venous blood; the former being supposed to be more stimulating than the latter.

2. To the more marked fibrous organization of the left valves and orifices, which renders them more liable to be converted into cartilage or bone.

3. To the lining membrane of the right cavities being continuous with the lining membrane of the veins, while that on the left side is continuous with that of the arteries; the latter being frequently the seat of adventitious deposit, which the former very rarely is.

4. To the more frequent obstructions to the circulation in the arterial than the venous system; but obstruction is common also in the pulmonary circulation.

5. To the greater stress upon the valves at the left than the right side of the heart, in consequence of the left ventricle having to transmit the blood throughout the system, while the right has merely to transmit it through the lungs.

Dr. Chevers\* is disposed to attribute "the greater immunity from mechanical injury and disease of the right heart, and pulmonary artery, in some measure to the comparatively yielding nature of their structures; the free reflux of which the tricuspid

\* On Morbid Conditions of the Pulmonary Artery.

lves admit whenever the ventricle is over distended, and the great pliability and extensibility of the muscular, valvular, and arterial tissues—circumstances which must all have an influence in rendering the parts less liable to mechanical injury, upon occasions of obstruction and over excitement.” “It is also highly probable (he thinks) that the cavities on this side of the heart are naturally more adapted to the reception of irritating fluids than are those on the left; as, in health, the former receive the highly carbonized blood from the veins, while the latter are accustomed to transmit only a perfectly depurated stream, so in disease, a strongly azotised or otherwise morbid fluid may be conveyed with impunity by the right cavities, while it produces the most irritating effects upon the left.”

#### EFFECTS OF VALVULAR DISEASE.

*The Primary effects* of any of the morbid conditions of the valves, which have been described, are to impede the free action of the valves, or to render them incapable of perfectly closing their respective orifices; to obstruct the current, to diminish the stream of blood passing through the orifices, or to render the surface over which it flows rough or irregular—by which the circulation through the heart is interfered with, or the ingress or egress of the blood is impeded or retarded.

When valvular disease has persisted for a period which varies in different cases, its *secondary effects* come to be developed, and we find some alteration in the chamber or chambers behind the affected valve in the course of the circulation, their parietes being either increased in thickness in order to overcome the impediment, or their walls yield to the distending force of the blood, and the tension of the chambers is increased. In general both results occur, and dilatation with hypertrophy ensues, and as the former or the latter predominates the symptoms and progress of the disease will be materially modified.

Thus when the semilunar valves of the aorta are diseased so as to contract the orifice, hypertrophy of the left ventricle sooner or later follows, and its cavity becomes also somewhat dilated, but hypertrophy predominates over the dilatation, and the circulation is maintained unless the contraction becomes extreme, or the patient's health breaks down. On the other hand, when the aortic

valves permit regurgitation, dilatation of the left ventricle sooner or later ensues, accompanied, in general, by some hypertrophy of its walls; but here dilatation usually predominates, owing to the state of distension in which the chamber of the ventricle is kept, particularly if the aperture permitting regurgitation is large, and the more dilatation predominates the less capable will the ventricle be of emptying itself; embarrassment to the circulation consequently supervenes earlier here than in simple contraction of the orifice, and if dilatation is in excess it may prove a cause of sudden death. “Over distension is known to be a cause of paralysis of the urinary bladder, and the diseased heart often ceases to act, not from any extension of the disease, but from a greater distension of its parietes having occurred than they could bear.”\*

When the mitral orifice is contracted, the cavity of the *left auricle* being kept in a state of over distension, becomes permanently dilated, while the chamber of the *left ventricle* not receiving its normal proportion of blood is sometimes diminished in size, and this may extend to the aorta, which in old cases is found to be smaller than natural—a point first noticed by Dr. Law.† The over distended state of the left auricle reacting upon the blood returning by the pulmonary veins, leads eventually to dilatation with hypertrophy of the *right ventricle*, followed by dilatation of the *right auricle*, and of its auriculo-ventricular aperture, and by *tricuspid regurgitation*, and impediment to the return of the venous blood from the system to the right side of the heart.

When the mitral valve permits regurgitation, dilatation of the *left auricle* follows, and if regurgitation is very free, it may react through the lungs upon the right side of the heart, just as in cases of contraction of the mitral orifice, but in a minor degree. Finally when the tricuspid orifice permits regurgitation, the cavity immediately behind it, that of the *right auricle*, becomes dilated, and the return of the venous blood from the system generally is impeded or retarded.

When both the aortic and mitral valves are diseased so as to obstruct the orifices, the cavity of the left ventricle may, Dr. Law† remarks, become diminished in size, and its walls be increased in thickness, constituting a variety of *concentric hyper-*

\* Furnivall on Dis. of the Heart.

‡ Dub. Quart. Jour. vol. xx.

† Dub. Jour. of Med. vol. xvii. 1840.

*ophy.* His researches show that this occurs under three conditions :

1. "When both the aortic and mitral valves are diseased."
2. "When the mitral valve is affected, while there is at the same time some distant obstruction in the course of the circulation."
3. "When there is a distinct obstruction, to which is superadded a diminution of the mass of the blood."

He considers it "to depend upon a law of the economy that the permanent capacity of the blood-vessels, whether of the cavities of the heart or the canals through which the blood flows, accommodates itself to the quantity of this fluid that is habitually present in them; and in obedience to this law these cavities and canals may either fall short of, or may exceed their normal dimensions."

We perceive then that the effects of disease of the aortic valves are expended principally upon the left ventricle—of the mitral valve upon the left auricle and the lungs, and through these organs upon the right side of the heart. That a state of the aortic valves permitting regurgitation is a more formidable lesion than a state of the mitral valve permitting regurgitation, inasmuch, as dilatation of the left ventricle which succeeds the former, causes more embarrassment to the general circulation than dilatation of the left auricle which succeeds the latter—and that a contracted state of the mitral orifice is a more formidable lesion than a contracted state of the aortic orifice, because in the latter the left ventricle becomes hypertrophied, and the circulation is maintained; while in the former, dilatation of the left auricle ensues, which reacting upon the lungs, and right side of the heart, leads to dilatation with hypertrophy of the right ventricle, and tricuspid regurgitation.

The *Tertiary effects* of valvular disease are exerted,

1st. Upon the *Lungs and bronchial mucous membrane*, giving rise to congestion, œdema of the pulmonary tissue, and pulmonary apoplexy; evidenced by dyspnœa, orthopnœa, cough, watery or acid expectoration, hæmoptysis, or hydrothorax.

2nd. Upon the *Liver* giving rise to congestion, and enlargement of this organ, interstitial deposit, and ultimately contraction and hardness; evidenced by deranged secretion, imperfect nutrition, dyspepsia, cachexia, hæmorrhoids, jaundice, or ascites.

3rd. Upon the *Gastro-intestinal mucous membrane*, evidenced

by nausea or vomiting, indigestion, colicky pains, flatulent distension, constipation sometimes alternating with diarrhoea, hæmatemesis, or intestinal hæmorrhage.

4th. Upon the *Kidneys*, evidenced by lumbar pains, by deranged, and diminished secretion of urine, by the presence of the red corpuscles or the albumen of the blood in this fluid, and by anasarca.

5th. Upon the *Heart* itself, by causing congestion of its tissue, or by impeding the coronary circulation, leading to softening or other degeneration, to laceration or rupture, or to the formation of polypiform concretions in its cavities; evidenced by feeble circulation, cold extremities, cardiac asthma, angina, failure of the powers of the heart, and sudden death.

6th. Upon the *Brain*, by disturbing the cerebral circulation in several ways, either impeding the return of the venous blood from this organ, diminishing the entire amount of arterial blood going to it, or cutting off the supply to a part of it, evidenced by headache, vertigo, sensations of throbbing or beating, ocular spectra, mental irritability, drowsiness or insomnia, disturbed sleep, delirium, coma, convulsions or paralysis.

7th. Upon *the general venous circulation*, evidenced by dropsy of the cellular tissue, and effusion into the serous cavities.

And, lastly, upon *the general health*, by deteriorating the blood, diminishing its globules, fibrin, and albumen, and increasing the proportion of the water; evidenced by anæmia, cachexia, debility, mucous hæmorrhages, and dropsy.

#### PROGNOSIS IN VALVULAR DISEASE.

The prognosis in valvular disease is influenced by a variety of circumstances, viz.—

1. The particular valve engaged, and the nature and amount of the structural change.

2. The length of time which the disease has lasted, and whether it has been stationary or progressive, or whether its progress has been rapid or the contrary.

3. The cause of the valvular disease, whether it has been the result of rheumatic endocarditis, or has supervened upon some other diseased state; or whether it has been the result of sudden rupture of a valve.



4. Its combination with morbid changes in other parts of the heart, as, with dilatation, attenuation, softening, or fatty degeneration of the ventricles, adherent pericardium, &c.

5. Its complication with anæmia, or other alteration of the blood, with renal disease, or dropsy.

6. The alterations consecutive to the valvular lesion in other organs, as the lungs, liver, kidneys, &c., and the amount and nature of these alterations.

7. The age, station in life, occupation and habits of the patient.

The valve engaged, the nature of the lesion and its degree or extent must exercise considerable influence upon the prognosis. The most formidable varieties of valvular disease are a state of the mitral valves permitting free regurgitation, and a very contracted state of the mitral orifice. A condition of the mitral valve permitting regurgitation is a less dangerous lesion than a condition of the aortic valves permitting regurgitation; and contraction of the mitral orifice is a less dangerous lesion than contraction of the aortic orifice, because it is not so soon followed by pulmonary, and general venous congestion. Indeed contraction is seldom met with in so great a degree at the aortic as at the mitral orifice, and it is in cases of the latter that the tertiary effects of valvular disease are usually most marked. Dr. Blakiston has given a table of 155 cases of disease of the heart, principally valvular, and he says there is hardly a single one in which death resulted from obstruction to either circulation, when the sole lesion was contraction of the mitral orifice. In the regurgitant lesion of the aortic valves, on the other hand, the left ventricle as a general rule becomes evenly dilated. When symptoms of angina appear in the latter, the prognosis is always more unfavourable; I have seldom known a patient labouring under this form of disease to live very long after the pain presenting the characters of angina became a prominent symptom, and the death was often sudden.

Valvular disease when uncomplicated, and moderate in degree, does not of itself necessarily shorten life, and many individuals, under these circumstances remove them from the necessity of labouring for their bread, live long, and eventually die of some other affection.

On the other hand, when valvular disease is complicated with disease of other parts of the heart, as adherent pericardium,

dilatation of the aorta, dilatation of the ventricles with attenuation or softening, the prognosis is unfavourable; when further complicated with congestion of the lungs, liver, or with dropsy, the chances of life are much diminished. The most frequent unfavourable complication of valvular disease is with dilatation of the ventricles, or with flabbiness or fatty degeneration of their tissue. Dr. M'Dowel,\* who has paid much attention to the subject, observes in reference to this point, "much variety is observed in the duration of different cases of valvular disease. Some run on to a fatal termination in a very short space of time; others on the contrary live for many years, suffering no doubt from various distressing symptoms, yet at intervals in the enjoyment of tolerable health. The difference in the amount of valvular disease in any two cases will rarely suffice to explain this circumstance; for in the case soonest fatal there may have been the least amount of valvular derangement. The accidental lesions to which the subjects of organic disease are so liable, as pneumonia, bronchitis, or fresh endocardial inflammation, will no doubt account for the fatal issue at an early period in many instances. But the great difference in the duration of life in all cases will be more obviously and more easily explained, if it shall appear that, in all organic diseases of the valves or apertures of the heart, the urgency of the symptoms is not so much proportional to the mechanical derangement, as to the changes produced thereby in the capacity, or the muscular development of the several cavities."

*The presence of disease in two valves at the same time, does not necessarily always make the case doubly serious, as supposed by some writers, nor even increase the danger; indeed it may in some instances have rather a compensating effect. Thus, in cases of aortic regurgitation, if the mitral valve likewise permits regurgitation, over distension of the left ventricle is not so liable to ensue, and dilatation of this chamber is retarded or prevented. The same I have found to occur when contraction of the aortic orifice is combined with contraction of the mitral; the diminution in the size of the latter prevents the blood from entering the left ventricle in greater amount than it can be readily transmitted from it, thus preventing over distension of the chamber.*

In some forms of valvular disease, however, the presence of

\* Dub. Quart. Jour. vol. xiv.

sease in both set of valves renders the case more serious. Thus, when regurgitant disease of the mitral orifice is combined with obstructive disease of the aortic orifice, embarrassment to the circulation is certain to ensue, because, there is not only a direct impediment to the passage of the blood into the aorta, but much of the strength of the ventricular systole is wasted, owing to a portion of the blood being transmitted backwards into the auricle at each ventricular systole.

*The loudness of a murmur* is by no means to be taken as an index of the gravity of the valvular lesion; the most formidable disease of a valve is sometimes unattended by any murmur, while a very loud bruit is sometimes the result of a comparatively harmless unsoundness. For instance, when the mitral valve imperfectly closes its orifice, and a small chink permits regurgitation a loud blowing, or musical murmur is often heard, while in the most advanced stage of disease of this valve, no murmur of any kind may be audible. Again a small vegetation projecting from one of the aortic valves, or a little mass of calcareous deposit upon the same part may give rise to a loud whistling or other musical murmur, which is sometimes audible even at a short distance from the patient, while a very contracted state of the same orifice may be unattended by any murmur.

*Complications of Valvular disease.*—Valvular disease is sometimes complicated with alterations in the normal constituents of the blood, as with anæmia, or plethora; the latter is most common in gouty subjects, and anæmia we know to be one of the results of protracted valvular disease, though it is sometimes also caused by depletory measures carried to too great a length. The forms of valvular disease in which the association with anæmia is most prejudicial are a state of the aortic valves permitting regurgitation, and a state of the mitral valve permitting regurgitation; in these it is certain to render palpitation and dyspnœa more distressing, and to add to the patient's suffering. Moreover, as the intensity of a valvular murmur is as a general rule increased by an anæmic state of the blood, it might in inexperienced hands lead to the employment of bleeding, or other antiphlogistic measures, which would be very certain to do harm. The prognosis in cases of this complication is influenced rather by the seat and nature of the valvular lesion than by the anæmia, the latter being more under the influence of remedies.

A deteriorated state of the blood is a more unfavourable complication, and is a frequent attendant upon valvular disease in its advanced stage. "The blood loaded with morbid matters resulting from bad digestion, or imperfect excretion, disorders (as Dr. Williams\* observes) the various organs through which it is distributed, and none more than the heart, whose injured structure makes it most susceptible of the disturbing influence; hence ensue attacks of palpitation, or irregular action and other symptoms of disturbance which magnify a disease before quiescent, into a distressing and possibly dangerous malady. Hence, we find the subject of organic disease of the heart occasionally, and in some instances almost periodically suffering from biliary or stomach disorders, from asthmatic or catarrhal affections, or from some of those multiform rheumatic, gouty, and kindred ailments that are connected with imperfect elimination by the kidneys."

*The Causes* which have given rise to the valvular disease are not without an influence upon the prognosis; thus valvular lesions the result of acute non-rheumatic endocarditis, are as a general rule more serious than lesions the result of rheumatic endocarditis, or of adventitious deposit in advanced life, because the injury the valves sustain at the time is almost always greater in the former than the latter. If endocarditis supervene in a patient the subject of Bright's disease of the kidneys, or whose health is broken down from other causes, if he does not succumb to the attack, the valvular lesion which remains is always very serious, and usually runs a rapid course. This is likewise observed when it depends upon the sudden rupture or laceration of a valve in an otherwise healthy subject; the parts apparently have not time to accommodate themselves to their altered condition; "nature, as Dr. Law observes, will submit to, and bear any amount of injury, if she have time to accommodate herself to it, but she cannot bear to be taken by surprise."

"The immediate danger in cases of this accident is very considerable, and death may supervene within a few days. Should however the patient rally from the immediate shock, and the subsequent inflammatory symptoms be overcome, life may be prolonged for a considerable period varying according to the nature of the injury, and the circumstances of the individual. Generally injuries

\* Lond. Jour. of Med. vol. ii.

the aortic valves, like affections of those valves dependent on ordinary causes, are more rapidly fatal than those of the mitral; and when the injury is such as to incapacitate them from closing the aperture, its effects are very much more serious than when obstruction to the flow of blood from the ventricle into the aorta only is occasioned.”\*

The alterations in other organs consecutive to the valvular lesion depend, as a general rule, upon the nature and amount of the impediment to the circulation occasioned by it. If the impediment is inconsiderable the disturbance to the circulation will be slight; if considerable, it will sooner or later be evidenced by congestion of the lungs, liver, and other organs; and ultimately by anasarca, and effusion into the serous cavities. The morbid condition of the valves in which these tertiary effects are most frequently witnessed, is contraction of the mitral orifice, followed by dilatation of the right chambers of the heart, and tricuspid regurgitation. Congestion when long continued, passes into structural alteration, the functions of the organ engaged are imperfectly performed, the digestion becomes deranged, the biliary secretion altered or suspended, the urinary secretion scanty, and the train of symptoms previously mentioned sets in, followed by anasarca, or effusion into the serous cavities, or by hæmoptysis and pulmonary apoplexy. Under such circumstances, the prognosis, it is scarcely necessary to say, is exceedingly unfavourable.

The occupation, station in life, and habits of the patient have considerable influence upon the progress of valvular disease. Thus, if the individual is removed from the necessity of labouring for his bread, and his circumstances enable him to take advantage of every auxiliary means for the preservation of health, the chances of life are much more favourable than under opposite circumstances. Hence, as a general rule the prognosis is less favourable in hospital than in private practice; the exposure to all the vicissitudes of the weather, to which the former in their occupation are perhaps unavoidably subject, with perhaps scanty food, insufficient clothing, and unhealthy habitation, tell unfavourably upon them; and if in addition, the habits, as is too often the case, are intemperate, the disease will undoubtedly run its course more rapidly.

The prognosis is influenced in some respects also by the age of

\* Dr. Peacock, Ed. Month. Jour. July, 1852.

the patient. When endocarditis occurs in infancy or childhood, and seriously injures the mitral or aortic valves, the progress of the disease is more rapid, and its entire duration is shorter than when it occurs at adult age; indeed such subjects seldom, and if they belong to the humbler classes, never survive much beyond puberty. On the other hand when valvular unsoundness, the result of rheumatic endocarditis in young subjects, is slight, and remains stationary for a considerable period, indicated by the murmur constantly preserving the same character, and when no sign of impediment to the circulation supervenes after several years, the prognosis is much more favourable.

There is however a source of danger in valvular disease independent of any of those enumerated; for instance, if an individual the subject of it, is attacked by another disease, the complication diminishes considerably his chance of recovery; and this applies not only to fever, and acute inflammatory affections generally, but, a comparatively slight affection, as influenza usually is, may, when occurring in the subject of chronic valvular disease, have an unfavourable termination simply from this complication.

#### TREATMENT OF VALVULAR DISEASE.

Organic disease of the valves once established is incurable, hence our treatment, so far as the lesion of the valve is concerned, can be only palliative, while as regards its effects upon other organs, it is mainly preventive. When valvular disease has passed beyond its first stage, our efforts are to be directed to the relief of the impediments to the circulation occasioned by it, and to the removal of its injurious effects upon the heart itself, the lungs, and other organs. In the more advanced stage, we can do little more than palliate the symptoms of obstructed circulation, remedy the derangements which are its result, relieve pain when an urgent symptom, and retard as long as possible the fatal termination. Still even in advanced cases, much may often be done for the patient's relief, provided his circumstances are such as admit of his conforming to the rules laid down, and he is willing to submit to the treatment considered necessary, and to attend to a number of minor circumstances which are indispensable in his condition.

The treatment of valvular disease is influenced in no inconsi-

erable degree by its stage; we have seen that the symptoms which characterize its several stages differ, and the treatment must be modified accordingly; it must be modified likewise by the seat and nature of the valvular lesion, by the particular symptoms which are most prominent, and by the age, habits, constitution, state of health, and other peculiarities connected with each case.

The indications of treatment in the first and second stages are—

1. To endeavour to remove the morbid condition of the valves, but, as that generally is not in our power, to prevent it from increasing.

2. To endeavour to prevent the supervention of dilatation of the ventricles, or if it has supervened to check further increase.

3. To relieve palpitation, to remove obstructions in the pulmonary or general venous circulation, and to diminish the quantity without deteriorating the quality of the blood.

4. To maintain as tranquil a state of the circulation as possible, to stimulate the heart's action when defective, to relieve it when embarrassed, to interdict all excessive muscular exertions, and to guard against errors in diet, exposure to cold, or intemperance.

When the valvular lesion is the result of rheumatic endocarditis, and is of recent origin, the treatment falls under that of chronic endocarditis; and its success will in a great measure depend upon the nature of the inflammatory exudation and its site, whether sub-endocardial, or upon the free surface of the membrane. Dr. Chevers thinks that we can distinguish between “fibrinous deposit in the sub-endocardial fibrous tissue, and lymph or fibrinous coagula upon the free endocardial surface of the orifice,” by the tone of the murmur; that heard in cases of sub-endocardial effusion being smooth and free, while in cases of deposit on the valves, whether in the shape of vegetations or otherwise, it is rougher, harsher, and sometimes musical. “We have strong grounds for the conviction, he adds, that fibrinous sub-endocardial deposits may, under favourable circumstances of constitution and treatment, become almost completely absorbed.” “Where, on the other hand, the obstructive material adheres to the endocardial surface, it may become slightly diminished in bulk, and smoothed and moulded by the continual passage of the blood, but the chance of absorption is small.”\*

\* Treatise on Diseases of the Heart, Calcutta, 1851.



In such cases the cautious administration of mercury will assist in promoting absorption of the morbid deposit; which should be given in moderate doses, and continued for several weeks if nothing contraindicates its use. Profuse or rapid salivation is not only unnecessary but hurtful, by deteriorating the general health, and inducing a cachectic state of the system. When valvular disease is of long standing, and there is no other indication for the exhibition of mercury, we should be slow in employing it, as, if it fail to do good, it seldom fails to do harm.

When this condition of the valvular apparatus is present, the patient cannot be too guarded in exposing himself to the influences which originally developed the disease; because when a valve has been once the seat of rheumatic inflammation, the latter is liable to recur (though in a less marked form) from exposure to causes which would have no deleterious influence in a healthy subject; and every fresh access of inflammation increases the valvular unsoundness, and adds considerably to the gravity of the lesion.

The seat of the valvular disease, whether in the aortic or mitral valve, its nature whether obstructive or regurgitant, and its amount or extent must be all taken into account. In fact, in some cases, little treatment beyond attention to the simplest hygienic rules is required; while in others we can do little more than postpone the occurrence of dilatation of the ventricles, with its train of evils. Thus, when mitral regurgitation is the result of rheumatic endocarditis, it occasionally happens that no other symptom is present than a bruit de soufflet, audible with the first sound of the heart; and the patient, for years subsequently, may be unconscious of anything wrong about his heart. Here the injury which the valve has suffered is evidently slight, and not sufficient to occasion impediment to the circulation; and if the patient's constitution is otherwise sound, if his habits are temperate, and his circumstances and position are favourable, he may suffer little inconvenience beyond being short-winded and incapable of making as great muscular exertion as before the rheumatic attack, and he may live as long as any other individual of the same age, unless he becomes the subject of some acute attack, when the valvular unsoundness will tell against him. On the other hand, if the seat of the endocarditis was at the aortic orifice, if it had had the effect of rendering the semilunar valves incompetent, and free regurgitation was per-



itted, increased action of the heart will be an early symptom, and no matter almost what treatment is adopted, dilatation of the cavity of the left ventricle will ensue, succeeded by other signs of impeded circulation.

To diminish the risk of dilatation, or if this has supervened, to prevent its increase, repose of the heart, as far as this is practicable, is essential—we thus prevent over distension of the cavities, and lessen the strain upon the injured valves; the organ is placed under favourable circumstances to adapt itself to the altered condition of the valves, and the process by which nature increases the propulsive power of the ventricles, and overcomes the impediment to the circulation, viz., strengthening and increasing the thickness of their parietes, is allowed free play. If, however, the valvular disease had remained stationary for several years, indicated by the murmur preserving the same characters as at first; and if during that period no signs of obstruction to the circulation had shown themselves, and the patient had been able to follow his occupation or profession, and to take exercise without palpitation or dyspnoea following, it would be unwise to make any alteration in his habits, or mode of life.

Palpitation generally becomes a prominent symptom when dilatation of the ventricles has supervened; it is constant, but is liable to be aggravated by trifling causes, and to occur in paroxysms of considerable intensity, accompanied by tumultuous action of the heart, pain in the præcordial region, dyspnoea and other distressing sensations. The treatment of the paroxysm will depend upon its cause, and is in some measure independent of the valvular lesion. Thus, when it arises from errors in diet, and derangement of the digestive organs, we often find flatulent distension to be the immediate exciting cause; and either constipation, diarrhoea, acidity, or nausea are also present; when the same means are to be had recourse to as where functional disturbance arises independent of valvular disease. When it depends upon an anæmic state of the system, it will be most effectually relieved by tonics in the intervals between the paroxysms, particularly the preparations of iron, among which the tincture of muriate of iron with bicarbonate of soda in a state of effervescence, the aromatic iron mixture, thetrate of iron and quinine, and the vinum ferri are perhaps the best. When, on the other hand, it appears to be the result of

plethora, or vascular fulness, it may be necessary to take blood from the arm: local bleeding is however in general to be preferred; and cupping between the scapulæ, or leeches to the præcordial region may be employed according to circumstances, combined with the internal administration of purgatives.

When valvular disease is accompanied by much constitutional irritability, with tumultuous action of the heart, oppression and dyspnœa, antispasmodics as camphor, sulphuric or chloric ether, Hoffman's anodyne liquor, ammonia, hydrocyanic acid, valerian or assafoetida variously combined, may be given. M. Lombard\* has found, camphor, assafoetida and polygala, the most effectual in relieving palpitation, in rendering the heart's action regular, and in removing dyspnœa. Dr. Munk† prefers tincture of digitalis in combination with "camphor, assafoetida or galbanum, ammonia or Hoffman's anodyne." The *depressing* effects of digitalis are produced, he observes, when the tincture is given per se, its *antispasmodic* effects when given in combination with the medicines above mentioned. "Thus employed, it will exert the most beneficial influence on palpitation, oppression, and distress in the præcordia, will relieve the hurry of breathing, and calm that irritability of body and of mind so generally witnessed under such circumstances." That this effect is owing to the digitalis rather than to the medicines with which it is associated, Dr. Munk has repeatedly assured himself by withdrawing the former and continuing the latter; "a loss in the amount of control over the heart has been the general result, and the converse of this has also been proved. Dr. Hope likewise recommended the tincture of digitalis in occasional doses in such cases, combined with some of the antispasmodic medicines before mentioned. M. Lombard prefers the powder of digitalis in combination with subcarbonate of iron, but according to Dr. Munk the powder is "the least certain, and most unmanageable of the preparations of digitalis." "I have not," he says, "succeeded by any combination in concentrating its action in a kindly manner upon the heart, and as an efficient and manageable sedative, I consider the powder of digitalis comparatively valueless." This remark does not however apply, he adds, to the combination of digitalis with mercury and squill, which "constitutes an admirable and efficient diuretic in cardiac dropsy." The tincture of aconite in

\* Gaz. Med. de Paris Oct. 1835.

† Guy's Hosp. Reports, Oct. 1844.

minute doses has sometimes a tranquillizing effect upon the heart, but it also often fails. In the paroxysm, a mustard cataplasm to the præcordial region, or immersing the feet in hot water to which some mustard is added, will contribute to relieve it.

The *sedative* influence of the tincture of digitalis upon the circulation, and the property it possesses of diminishing the frequency of the heart's action have led to its too indiscriminate use in valvular disease. There are no objections to occasional doses, in combination with the subcarbonate or aromatic spirit of ammonia, or ether, with camphor mixture, but there are strong objections to its continuous employment until an effect is produced upon the circulation; there is scarcely any form of valvular disease, in which thus employed it can be of use, and there are some in which it is decidedly dangerous. Even in its mildest form, that of infusion, when used as a diuretic in cardiac dropsy it is, Dr. Kilgour\* says, "a dangerous medicine, and not unfrequently the immediate cause of death, the patient going off in a convulsion brought on by this, to him, poisonous drug."

The general effect of valvular disease we have seen to be to impede the circulation, leading in many instances to dilatation of the chamber behind the affected valve in the course of the circulation, by which its power of overcoming the impediment to the circulation is diminished; and if, in such a case, we diminish the frequency of the heart's action by administering digitalis, its power of maintaining the circulation will be lessened in proportion. Again when obstruction and retardation of the circulation have supervened in valvular disease, and their effects are visible in the lungs, and general venous system, the action of the heart is often strong, while the pulse is feeble, and the extremities are cold; showing that notwithstanding its increased action, the blood is imperfectly propelled through the arterial system. In such cases, digitalis continued until it produces a sedative effect upon the circulation is attended with considerable risk.

Dr. Corrigan† was the first to call attention to the dangers attending the administration of digitalis in patency of the aortic valves: "The danger of the disease is in proportion (he observes) to the quantity of blood that regurgitates; and the quantity that regurgitates will be large in proportion to the degree of inadequacy

\* Ed. Month. Jour. Sep. 1843.

† Ed. Med. and Surg. Jour. vol. xxxvii. 1832.

of the valves, and to the length of pause between the contractions of the ventricles. If the action of the heart be rendered very slow, the pause after each contraction will be long, and consequently the regurgitation of blood must be considerable. Frequent action of the heart, on the contrary, makes the pause after each contraction short. Instead, then, of regarding an increase of frequency in the action of the heart as an aggravation of the disease, it must be viewed as a provision for remedying as far as possible, the evil consequences arising from inadequate valves. To retard in such circumstances the action of the heart would be to do an injury. In every case of this disease in which digitalis has been administered, it has invariably aggravated the patient's sufferings. The oppression has become greater; the action of the heart more laboured; the pulse intermittent, and very often dicrotic, from the heart's being unable by a single contraction to empty itself; general congestion and dropsy, if present have been increased."

It was at one time supposed that hypertrophy constituted a very serious complication of valvular disease, and the efforts of physicians were directed to the diminution or removal of this state, which they endeavoured to accomplish by bleeding, purgatives, and low diet. But we now know, that hypertrophy succeeding to valvular disease, is nature's provision for overcoming the impediment to the circulation occasioned by the valvular lesion; and that the lowering treatment employed to remove this state is likely to prove more injurious than useful. Dr. Corrigan\* was, I believe, the first to call attention to this point: "Too often in the treatment of a valvular alteration in the heart, there has been (he observes) a constant struggle between nature and medicine. Nature has been making the organ equal to its task, while medicine has been directed to counteract nature's efforts, and by weakening the organ, to render it totally incapable of its task. The repeated bleedings, the starvings, the enforcement of debilitating measures, are totally unsuited to the disease we are considering. Instead of such treatment, the measures most beneficial are those which by strengthening the general constitution, will give a proportionate degree of vigour to the muscular power of the heart, and thus enable it to carry on the circulation."

If any medicine possessed the power of acting directly as a

\* Ed. Med. and Surg. Jour. vol. xxxvii. 1832.

upon the heart, it would be a valuable addition to our other means in cases such as these. Recently, nux vomica and its alkaloid, strychnia, have been recommended with this object, and several physicians have reported favourably of them; indeed Dr. Brown\* terms strychnia "*the tonic of the heart.*" It should be given, he says, in very minute doses, long-continued, viz. from the twentieth to the fortieth of a grain, and be persevered in for weeks or months. "It gives tone to the faltering organ, and especially cures atonic dyspepsia, often so troublesome, and gives the patient new appetite and spirits." "The most convenient form, according to him, is either a solution of one grain of strychnia in six drachms of water, with two drachms of acetic acid, or what is sometimes better in dyspepsia, from its vegetable bitter, a strong decoction of nux vomica. Either may be given alone in doses of five to fifteen drops in water, three times a day; or they may be combined with preparations of iron, or other tonics." Dr. Law† is favourably of a combination of James' powder and extract of nux vomica, in certain cases of valvular disease with cerebral debility. "The former ingredient by determining to the surface produces an equalization of the circulation, while the latter stimulates the heart as a muscular organ."

The indications of treatment in the tertiary stage of valvular disease are:

1. To diminish or remove congestion of the lungs, liver, &c., and the general venous circulation.
2. To relieve cough, dyspnoea, or pain, when prominent symptoms.
3. To endeavour to improve the condition of the blood, when deficient in any of its essential constituents.
4. To correct and promote the various secretions, particularly of the kidneys, liver, intestines, and skin.
5. To prevent the occurrence of dropsy, or if it has supervened, to relieve or remove it without debilitating the patient.

When valvular disease has reached its tertiary stage, dyspnoea and cough are the most prominent symptoms in one class of cases; while in another, pain having the character of angina, constitutes the chief source of suffering. In the former, more or less pulmonary congestion is generally present, and the lesion has usually its

seat at the mitral orifice, which is often contracted; in the latter the aortic orifice is the usual seat of the lesion, and these valves will be found to permit regurgitation.

In congestion of the lungs, the result of valvular disease, general blood-letting to a small amount is occasionally of use, its employment however requires caution, and it should never be carried so far as to produce syncope. Local is more frequently employed than general bleeding, and cupping or leeches may be used according to circumstances. A good situation for the cupping glasses is between the scapulæ, and if the patient is too weak to bear any loss of blood, dry cupping over the front or back of the chest may be substituted, taking care not to allow the cupping glasses to remain too long upon the part. After bleeding, or independent of it, counter irritation is a valuable adjunct, and the ordinary blister is perhaps the best, repeated according to circumstances. Purgatives by increasing the intestinal secretions, frequently assist in diminishing pulmonary congestion, while they at the same time are useful by removing morbid matters from the system, and when well borne they give decided relief. Expectorants are also of service, and relief to the dyspnoea generally follows free expectoration; the preparations of ipecacuanha, squill, antimony and ammoniacum may be employed for this purpose, they are advantageously combined with camphor, ether, or ammonia, and they may be alternated or further combined with alkalies or diuretics.

When *pain*, having the character of *angina*, is a prominent symptom, relief can generally only be obtained from narcotics, which are advantageously combined with antispasmodics. The salts of morphia particularly the muriate are highly useful in these cases, but the preparation of opium from which I have derived most benefit, and which can be persevered in for the longest period, is the liquor opii sedativus; a good combination for it is with sulphuric ether and camphor mixture, according to the formula given by Dr. Law, though some patients object to the ether, and it is often equally efficacious without it. In some instances chloroform in the dose of twenty drops gives relief for a time; in others aconite, belladonna, or chloroform applied externally afford temporary relief; in others, mustard cataplasms or blisters over the seat of pain, or a seton in the præcordial region are of service; M. Rombe

one instance found nitrate of silver, in small doses, of temporary use.

The advanced stage of valvular disease is very generally accompanied by disorder of the digestive organs; the patient suffers from flatulence, acidity, distension, or pain, by which palpitation and dyspnoea are aggravated; associated with which, or independent of them the stomach is sometimes very irritable, or diarrhoea is troublesome. The special treatment of these complications does not however present any peculiarity. I would merely remark, that an irritable state of the stomach and bowels may sometimes be remedied to the exhibition of nauseating expectorants, or hydragogue cathartics; we ought therefore to be guarded in the employment of such medicines in the advanced stage of valvular disease; indeed when either vomiting or diarrhoea ensue, as the result of the congested state of the gastro-intestinal mucous membrane, they constitute most disagreeable and unfavourable complications, limiting considerably in the number of our remedies, and by the debility they occasion, hastening the fatal termination of the disease.

When *hepatic congestion* is present, indicated by enlargement of the liver, pain or uneasiness in the right hypochondrium, or epigastrium, and deranged biliary secretion, benefit will be derived from small doses of mercury in combination with taraxacum and extract of rhubarb, given two or three times a day, and continued until a slight effect is produced upon the gums. Or a mercurial purgative may be given at night, followed by a saline in combination with an alkali in the morning, and repeated according to circumstances. At the same time, cupping or leeches over the enlarged organ are generally necessary, followed by counter-irritation. In protracted valvular disease, the blood, as has already been observed, undergoes material alterations; indeed some of its tertiary effects are directly due to these alterations; thus the red corpuscles are diminished, and the watery parts are increased, by which œmia is induced; the fibrin is also diminished, leading to hæmorrhage from mucous surfaces: and, the albumen of the serum of the blood usually undergoes diminution preliminary to the occurrence of dropsy. The morbid conditions of this fluid should therefore equally engage attention, and treatment calculated to improve the quality of the blood, and through it the general health, often constitutes the most useful general means. Dr. S.



urinary secretion. The cases to which it is best adapted in which the valvular disease is associated with anæmia, or where slight renal complication exists, and rather than mitral valve disease.

Stimulants are generally necessary at this period, and be combined or alternated with any of the classes of medicine mentioned. Dr. Williams† speaks favourably of the potassium as a stimulant in cases of this kind, in doses of from five to fifteen grains, combined with carbonate of ammonia, and given at short intervals.

In the treatment of protracted valvular disease, the physician should however avoid the error of endeavouring to do too much by medicine; if it is not called for by the symptoms, it should not be given. Our objects can often be more safely and effectually attained by rest, diet, and such means, than by the exhibition of drugs. Indeed, I think with Dr. Corbly, "protracted valvular disease is commonly over-treated," and as a result, the stomach is deranged or rendered irritable, the bowels are disordered, and palpitation aggravated. "It is a golden rule in medicine, and especially in these affections, (he says) when *nothing is to be done, to do nothing.*"

The importance of *rest* as an auxiliary to the treatment in the advanced stages of valvular disease cannot be too much insisted on; all unnecessary, and in some cases every kind of

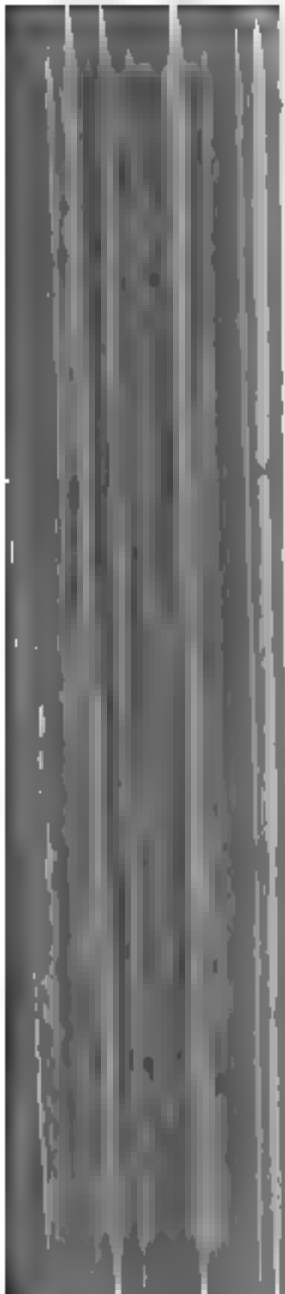


the general health, and increasing nervous irritability. Here, exercise, either active or passive, suited to the previous habits of the patient should be regularly taken, and when circumstances permit of it, travelling, change of air and of scene, are advantageously combined. For these reasons, the treatment of patients among the better classes is much more satisfactory than with hospital patients, whose daily bread often depends upon their returning to their employment immediately on leaving hospital. Strong mental emotion of every kind ought to be avoided by the subjects of valvular disease in its advanced stage, more particularly in aortic regurgitant disease; indeed violent excitement, as a paroxysm of anger has proved suddenly fatal in this form of disease.

The *diet* in the advanced stage of valvular disease should be nutritious, the amount of solid food taken at a meal not so great as to occasion distension, and its kind such as is of easy digestion, and not likely to occasion flatulence. Much however must depend upon the habits and constitution of the patient, and no rules applicable to all cases can be laid down, as what suits one subject may be ill adapted for another. *Liquids* being absorbed rapidly and finding their way quickly into the circulation should be sparingly used; indeed embarrassment to the circulation is very likely to be increased by their too free use. One of the most effectual means we possess of diminishing the mass of circulating fluid and thereby lessening vascular fulness, without impoverishing the blood, or debilitating the system, consists in limiting the patient to as small an amount of liquid as is compatible with health. Every particle of watery fluid swallowed must pass through the circulation before being excreted by the kidneys, or skin; and as, in the cases we are considering, an obstacle often exists to the free passage of the blood through or out of the chambers of the heart, if the mass of blood is increased by the ingestion of a quantity of watery fluid, the heart will have still more difficulty in propelling its contents, and increase of distress will inevitably ensue. Hence the necessity in this class of cases of what has been termed a "*dry diet*," that is of a diet consisting of a very moderate allowance of liquid.

## TREATMENT OF THE DROPSY.

When dropsy supervenes, valvular disease may be said to have reached its ultimate stage, which might not inappropriately be



appearance, and in general it is at first amenable more so perhaps than the dropsy which supervenes on organic diseases; it may even recur several times after yield to treatment, but eventually a period arrives when the remedies fail to act, the anasarca goes on increasing despite the treatment, is superadded, followed by hydrothorax, and the patient enters a protracted agony. As long as the blood is not discoloured, the muscular tissue of the heart not weakened, the urine merely diminished, and the vital powers but little depressed, will the treatment of this form of dropsy prove satisfactory. We have often patients in hospital, who, after remaining some time out relieved as far as the dropsy is concerned; a fresh swelling brings them again to seek admission, and they are admitted several times. At length with the recurrence of the dropsy, with its gradual increase, the urinary secretion becomes scanty and affords an abundant deposit of lithates, or in the presence of albumen, the remedies formerly of use become inoperative, or no longer produce any effect, and the patient enters a more or less protracted struggle.

The remedy, or rather the combination upon which we usually rely on the first appearance of dropsy, and perhaps the greatest amount of benefit is derived, is blue pill with digitalis and squill, continued until a slight effusion is effected upon the gums. Cardiac dropsy we have said is preceded by pulmonary and hepatic congestion, and it seems to exert a salutary influence at this period by

the bowels should be unloaded, and it is usually advisable to follow it up by tonics. Even when the dropsy has lasted some time, if the patient's constitution is otherwise good, this plan of treatment proves often effectual, and may be resorted to more than once, if a return of the dropsy calls for a repetition of treatment. When however the disease is more advanced, and the patient's health is broken down, and particularly when renal is superadded to cardiac dropsy and the urine is albuminous, and of low specific gravity, a mercurial course, however mild, would be sure to aggravate instead of relieving the disease, and to hurry it to a fatal termination.

The other remedies upon which reliance is placed at this period, in cases to which they are adapted, are hydragogue cathartics, and diuretics. It must be borne in mind, however, that "when it is considered necessary to excite the action of congested kidneys and intestines, it is absolutely useless and unsafe (as Dr. Chevers remarks) to attempt to bring the two organs into operation at once. If direct diuretics or strong purgatives are to be given, the administration of each should form a separate plan of treatment. If under the employment of means tending to relieve congestion one organ recovers its power of acting, it is as much as we can expect; and its relief will probably conduce to the disembarrassment of the other. On the contrary, in attempting to excite both with a precarious chance of success, a double strain is laid upon the system, and a double prospect of failure is entailed."

*Hydragogue Cathartics*, in cases to which they are adapted, sometimes afford much temporary relief by removing a quantity of watery fluid from the system, diminishing pulmonary or general venous congestion, and relieving the dyspnoea and cough. The most energetic we possess is elaterium, and its effects are sometimes surprising, evacuating a very large quantity of liquid by stool within a short period. It has the disadvantage however of causing nausea, depression and exhaustion, it should never therefore be given if the patient is either very young or advanced in life, if the heart is weak and dilated, or the vital powers are low; and, the patient's strength must be supported during its administration by wine, and other stimulants. Even under the most favourable circumstances, it is not a medicine to which we can often have recourse, and less frequently now I think than formerly, while its employment always requires caution. Elaterium is best given in the form of pill, in

combination with creasote, and extract of hyoscyamus, by which its tendency to cause nausea and vomiting is diminished; though Dr. Ferriar,\* to whom we are indebted for recalling attention to this medicine in dropsy, always administered it in the fluid form. The dose should never at first exceed the twelfth or the eighth of a grain. I have seen hypercatharsis produced by the fourth of a grain. The late Dr. John Crampton† was in the habit of employing the powdered leaves, and the fruit of the plant, which he says possess all the sensible properties of the drug, and are much milder.

The compound powder of jalap is a safer medicine than elaterium, and less likely to cause nausea, vomiting, or depression. The bitartrate of potass in the dose of from two drachms to half an ounce twice a day, sometimes proves an excellent hydragogue cathartic, and in addition usually increases the urinary secretion. It may be given in the form of electuary, and its operation may be quickened if necessary by the addition of a few grains of jalap to each dose. It has the disadvantage however of being uncertain, sometimes causing excessive evacuations, sometimes appearing to be almost inert.

If *Diuretics* were as certain in their operation as cathartics, and if we could always depend upon their increasing the urinary secretion, they would constitute the most valuable agents in cardiac dropsy, by removing watery fluid without debilitating the patient. This however is far from being the case, their operation is capricious, and often uncertain. The cause of this often lies in their not being preceded by measures calculated to relieve or remove renal congestion, and vascular fulness. In the kind of cases we are considering, where in addition to general venous congestion the kidneys themselves are often in a state of hyperæmia, it is vain to expect to force an increase of the urinary secretion by the administration of diuretics, the congestion must be relieved before they can act. This may be accomplished by cupping over the region of the kidneys, or when pulmonary congestion is marked by a small bleeding from the arm, as originally recommended by Dr. John Crampton,‡ and more recently by Sir Henry Marsh. “In obstructive cardiac disease, the dropsy often increases (k

\* Medical Histories, vol. iv, 1810.

† Trans. of Col. of Phys. vol. ii. 1871.

‡ Trans. of Assoc. of Col. of Phys. vol. ii.

§ Dub. Quart. Jour. vol. xvi.

erves) despite the most powerful diuretic and cathartic medicines; one small bleeding from the arm suffices to alter the whole condition of the vascular system. The congestive tension of the vessels is removed, immediately the absorbents work, and the diuresis becomes profuse."

The number of medicines belonging to the class of diuretics is considerable and almost every practitioner has his favourite formula. The diuretics which appear to be most useful in cardiac dropsy, in the order of their efficacy are—

Nitrate of potass; infusion of digitalis; acetate of potass; tincture of cantharides; gin and water; sweet spirits of nitre; infusion of broom; and water of acetate of ammonia.

We generally derive more advantage from a combination of medicines belonging to this class than from any one singly, and we often find it necessary to change the formula in the same case. In many instances, tonics are advantageously combined with diuretics, and sometimes tonics, particularly iron and quinine, prove the best diuretics.

*Digitaline* in minute doses (the 75th of a grain), has recently been recommended as a substitute for digitalis by M. M. Homolle and Quevenne. The advantages it possesses over the infusion of digitalis according to them, are that it is less likely to derange the stomach, and, if the latter is irritable, it is better borne. Dr. Christison\* who has given it a fair trial, says, that in his hands it has proved equally or more serviceable in dropsy connected with disease of the heart than in other forms. It is liable however occasionally to induce sedative, rather than diuretic effects.

The states which are incompatible with digitalis, or any of its preparations, are, as Dr. Munk† has shown, gastric irritation and inflammation, and a plethoric condition of the system. "Digitalis seldom (he observes) operates as a diuretic when its action on the heart is prominently marked, and conversely, it seldom manifests its action on the heart when free diuresis results from its employment." He is in the habit of prescribing it "in the usual doses for a week, and if, within that period, he perceives neither sedative or diuretic effects, he invariably desists from its administration." "Let these effects however be once (he adds) kindly

\* Ed. Month. Jour. Jan. 1855.

† Guy's Hosp. Reports, Oct. 1844.

induced, and the medicine may then be continued with safety for a considerable period."

*Diaphoretics* are sometimes useful substitutes, where diuretics disagree; Dr. Chevers speaks favourably of them, and Dr. Graves advocated their use in cardiac as well as in other forms of dropsy, provided diaphoresis can be easily induced.

Our efforts for the removal of the dropsy are not, it is scarcely necessary to say, always crowned with success, it increases despite of remedies, the lower extremities attain sometimes an enormous size, and the scrotum and penis, or the labiæ in the female become also greatly swollen; the patient suffers extreme pain from the distension, and is reduced to a most helpless, and pitiable condition. At this period, the skin sometimes cracks, and serum escapes with temporary relief, or erythematous patches appear upon the legs followed by vesicles which discharge serum in abundance, or large bullæ form upon the dorsum of the foot, which break, and allow of the escape of the fluid. When the scrotum and penis, or the labiæ are considerably swollen, the urine can scarcely be prevented from coming in contact with the anasarcaous parts, which it irritates, and inflames, the cuticle separates and gangrene follows.

When the distension is extreme, when the patient is suffering excessive pain in consequence, and when from the appearance of the parts gangrene seems to be imminent, temporary relief may be afforded by acupuncture of the anasarcaous limbs; it is however a dangerous remedy, particularly if the punctures are made below the knees; I have seen it quickly followed by erysipelatous inflammation, and gangrene, with excessive suffering. This is less likely to ensue if the punctures are made in the upper part of the thighs, as proposed by Dr. Watson; but at best it is an unsafe practice, and independent of gangrene, other disagreeable results may ensue. Sir H. Marsh alludes to cases when "from the perpetual oozing of cold serum, the broken down state of the health, and of the blood, each puncture has been followed by a large and foul ulcer. "In other instances of hopeless dropsy, even though the patient escape these grave consequences of acupuncture, the limbs have been kept so constantly wet and cold as greatly to distress the patient, and aggravate his sufferings."\*

A safer proceeding than acupuncture, and one less likely to

\* Dub. Quart. Jour. vol. xvi.

followed by erysipelas, or gangrene, is the old method mentioned by Freind,\* approved of by Mead,† and revived by M. Lombard,‡ which consists in making *incisions* in the anasarctous lower extremities. Mead recommended an incision on the inside of the leg, about two inches above the ankle, not deeper than the cellular tissue; the part to be fomented afterwards, and the patient's strength to be supported at the same time. M. Lombard makes four or five incisions in each leg down to the fascia, the third of an inch in length in the most depending position; and when the serum has all drained away, he supports the limbs by a roller. He advises that the incisions be made earlier than is usually done, as when the skin has been long distended it is liable to slough independent of either punctures or incisions. Dr. Kilgour,§ who is also in favour of this practice, objects to early incisions, because the fluid does not flow so freely as when the areolar tissue has become more open from the long continued pressure of the effused serum.

The numerous incisions advocated by M. Lombard are unnecessary, and in several respects objectionable; one in each limb ample, if it be deemed advisable to have recourse to them. I have been content myself with a single incision in one leg; it is not essential either to cut down to the fascia, and the incision could be made in the middle or upper third of the leg, taking as a precaution to avoid wounding a vein, supporting the patient's strength subsequently, and keeping the limb as dry and warm as possible by wrapping it in warm cloths, which as long as serum continues to escape should be frequently changed. Dr. Home|| informs us that he has used issues with success for this purpose, and he prefers them to incisions; they were made in each leg below the knee, and three cases are given where the removal of the dropsy followed their employment. I am not aware that this method has had any advocate since.

\* Historia Medicinæ.

† Monita et Præcepta Med. 1757.

‡ Gazette des Hôpitaux.

§ Ed. Month. Jour. vol. ii. 1850.

|| Clin. Exper. and Hist. 1780.

## CHAPTER XVI.

### MORBID CONDITIONS OF THE PARIETES AND CAVITIES OF THE HEART.

#### MYOCARDITIS, CARDITIS. — ANATOMICAL CHARACTERS. — SYMPTOMS. — EFFECTS OF.—CAUSES AND PROGNOSIS.—CONCLUSIONS RESPECTING.

ACUTE inflammation of the muscular tissue of the heart except in association with pericarditis, or endocarditis, is very uncommon, and even associated with inflammation of these membranes it is rare. When endo-pericarditis occurs in an intense form, the inflammation may extend to the muscular fibres in contact with the inflamed membranes, and this, although it cannot strictly be termed myocarditis, constitutes almost the only form of the disease with which we are acquainted. The inflammation generally extends but a short depth into the muscular substance of the heart from the pericardial or endocardial surfaces; it may however engage a *portion* of the parietes in their entire thickness. It is in a great measure confined to the parietes of the left ventricle, and in general to a limited portion of it.

#### ANATOMICAL CHARACTERS.

Three stages of the disease are distinguished by continental pathologists—

The first consists in “an injected condition of the minute vessels which in a dense network traverse the cellular tissue situate between the fibres; the heart’s walls being darker coloured, and firmer than natural.”\*

In the second (that of softening) exudation of serum, either limpid or coloured with the red particles of the blood takes place into the cellular tissue between the fibres. “The muscular fibres lose their consistence through maceration in this their degenerate nutrient web; they lie in it as it were isolated, and assume according to the peculiarity of the inflammatory product a pale,

\* Hasse Pathological Anatomy.



dingy-gray, yellowish, or dirty brown hue ;” \* occasionally a very dark colour, and the tissue becomes soft, loose, and friable.

The third stage, or that of suppuration, is characterized by still further softening, and by the formation of pus, which is either infiltrated in the tissue, or collected into small abscesses. The latter are almost limited to the walls of the left ventricle ; and the pus may have the ordinary colour, or be tinged by the colouring matter of the blood. According to Rokitansky, *induration* is a more common result of myocarditis than suppuration. But induration, in other situations, is rather the result of chronic than of acute inflammation.

When abscess forms in the tissue of the heart, the matter may ultimately make its way by ulceration, or rupture to the endocardial or pericardial surface ; in the former giving rise to pyæmia from admixture of pus with the blood ; in the latter to pericarditis, which is quickly fatal. When the septum of the ventricles has been the seat of abscess, and perforation has occurred, a permanent communication between the ventricles has been established. Abscess in the muscular tissue of the heart arising from purulent infection of the blood, is much more common than abscess the result of myocarditis ; and, might be mistaken for it. They are distinguished by the previous history of the patient's illness, and by the condition of the surrounding tissues.

#### SYMPTOMS OF MYOCARDITIS.

Myocarditis scarcely ever, as I have said, is met with as a solitary disease, being always associated with pericarditis, or endocarditis, sometimes in addition with pneumonia, or pleuritis ; even in association with them it is very rare, and its symptoms are a combination of those of intense endo. and pericarditis.

According to Dr. Copland, † the presence of the following train of symptoms would lead to a suspicion of the existence of acute carditis. “ The patient experiences a violent pain in the region of the heart, with anxiety, preceded or attended by rigors, chills, or tremblings of the whole frame. To these succeed increased heat about the præcordia, or in the trunk, while the extremities and face are cold, and the whole surface is covered by perspiration, which is cold on the extremities. The pain is concentrated in the

\* Hasse Pathological Anatomy.

† Dict. of Medicine, vol. ii.

situation of the heart, is lacerating, or rending, accompanied by the utmost agitation, and expression of anxiety, and distress, sometimes by screams, and occasionally by general convulsions and swooning. The patient feels every pulsation of the heart, rolls about to obtain ease, and presses his hand forcibly against the præcordia. The chest is elevated, the head thrown back, and the face and hands covered with cold sweat. There is great thirst, but drink is refused on reaching the lips; and, there is often loquacity passing into delirium as the disease advances." . . . "The pulse varies remarkably, but is generally unequal, or irregular, and remarkably small, and weak, or indistinct. There is neither cough, nor expectoration, nor vomiting, but a frequent expression of pain, and distress." . . . "The seizure is generally sudden, and the disease reaches its acmé about the third day." Dr. Hope was of opinion that carditis might be "the cause of the feeble, fluttering, irregular, intermittent action of the heart, with suffocative symptoms, when these phenomena could not be accounted for by the presence of fluid in the pericardium, or of polypous concretions from endocarditis."

None of the foregoing symptoms can however be said to be pathognomonic; they have all been observed in intense endocarditis, particularly where the inflammation extended beyond these membranes, and the pleura covering the diaphragm became engaged. If, however, such a combination of symptoms should present, if they supervene during the course of acute rheumatism, and if the physical signs of pericarditis or endocarditis, are present, the probabilities are in favour of carditis, implicating the layer of muscular fibre in contact with the inflamed membrane. The following case recorded by Dr. Stokes\* affords an example.

"A youth, aged about eighteen, after being excited, and heated by violent gymnastic exercise, slept for several hours on his left side on the damp grass; he awoke in a state of distress attended by pain in the præcordial region so severe as to prevent him from lying down. More than a week elapsed before I was called to him. On my first visit he presented all the symptoms and signs of the most violent pericarditis, and this condition, though somewhat mitigated, remained until the patient's death. No opium seemed to have the slightest effect in controlling the dis-

\* Treatise on Diseases of the Heart.

ent suffered in an exaggerated form all the miseries of a violent liac inflammation, and had the most indescribable and permanent anguish. On dissection, the pericardium contained a quantity of coffee-coloured, sanious fluid, mixed with shreds of coagulable lymph. The serous membrane was covered thickly by a dark-coloured false membrane, so disposed as to give a generally honeycombed appearance to the entire surface of the heart. In numberless points ulcerative absorption of the serous membrane had taken place, and corresponding to these were well-defined depressions in the muscular structure of two or three lines depth, and of the same, or even a greater extent in diameter, evidently resulting from loss of substance in the muscle itself. The whole heart had a livid, almost black hue, which however increased in intensity as we approached the inner layers, and *columnæ carneæ*."

A case recorded by Mr. Salter,\* is remarkable for the absence of all signs of peri. or endocarditis. The patient, a man aged fifty, suffered from oppression, dyspnœa, and orthopnœa, with dull heavy pain at the sternum; the pulse was eighty and regular, and the heart's action natural; he died after a short illness. On examination, the pericardium and endocardium presented nothing abnormal; the heart was somewhat larger than natural, its substance moderately firm. The muscular parietes of the left ventricle, except near either surface, were of a lightish yellow hue, and from the cut surfaces purulent matter could be scraped. In some parts of the muscular substance little cavities were found, from the size of a pin's head, to that of a small pea, and filled with pus.

In a third well-marked example, no symptom whatever was present during life to attract attention to the heart, and the illness, which lasted four days, was supposed to be cerebral inflammation. The case is recorded by Mr. Stanley,† and was witnessed by Dr. Graham, who has given the following summary‡ of its leading features.

"A boy, twelve years of age, was in perfect health on Saturday night, and dead on the following Tuesday afternoon. He had, in the opinion of all who saw him, the severest inflammation of the brain. The attack was sudden, with great heat and frequency of

\* Med. Chir. Trans. vol. xxii.

† Med. Chir. Trans. 1816.

‡ Lectures on the Heart, vol. ii.

the pulse. He had delirium and convulsions, and point of forehead as the seat of the pain. At length he sank into insensibility and died. Upon dissection not a vestige of inflammation was found within the cranium, but the heart was the seat of most intense inflammation pervading both the pericardium and the muscular substance. Four or five ounces of turbid serum and flakes of lymph floating in it, were found in the cavity of the pericardium, which had its internal surface covered in various places with a thin layer of reticulated lymph. There was no communication between the two surfaces; the lymph and the serum had effused together, and the serum had partially washed away the lymph as it was deposited. Further, when the heart was divided, the muscular fibres were dark-coloured almost to black, loaded with blood, soft and loose of texture, easily separated and easily torn by the fingers; and at the cut edges of *both* ventricles small quantities of dark-coloured pus were seen among the muscular fibres. The internal lining was of a deep red colour from any effusion of lymph."

In myocarditis the pus instead of being diffused through the tissue of the heart may be collected into little abscesses, when the inflammation had been limited, or when it has assumed a more chronic form. The parietes of the left ventricle are the most frequent seat of these abscesses; they are usually single, but several have been met with in the same subject. They are generally small, viz: from the size of a pea, to that of a walnut. Graves has however recorded a case where the sac was containing more than two ounces of pus; and they are lined by a pyogenic membrane.

Abscess of the heart is sometimes preceded by signs of pericarditis, at others nothing of the kind is observed. In Graves' case, to which I have alluded, the patient a man of fifty-five years of age, had laboured under dyspnoea, and cough for many months, followed by anasarca. As related by Dr. Graves he "suffered much from distress and pain in the region of the heart. This pain formed the chief of his complaint, and darted over the chest." "A loud and harsh bruit de soufflet existed together with a remarkable frequency of the pulse, and a very irregular pulse." Signs of hypertrophy

\* Clinical Lectures, vol. ii.

dilatation of both ventricles were likewise present. The pain in the region of the heart continued, and was at times excruciating, and the patient died at the end of a few weeks.

On examination, the pericardium was adherent: the adhesions were easily broken down except at the apex of the heart, where they were strong and firm; "in attempting to break them more than two ounces of purulent matter escaped;" and on further examination a "small rent was found at the apex of the heart, in the situation of the firm adhesions, on enlarging it a cavity in the substance of the heart was brought into view, having a regularly defined wall, and capable of containing more than two ounces of fluid. The walls of both ventricles were enormously thickened; all the valves were more or less affected, but the chief disease lay in the semilunar valves of the aorta, which were nearly altogether ossified."

The cases in which symptoms of myocarditis were absent are, however, the most numerous, two such have been placed on record within the last few years, one by Mr. Chance,\* the other by Mr. Howitt.† In Mr. Chance's case, the patient a boy of thirteen years of age, and previously in good health, was attacked with vomiting, followed by collapse, and died comatose the following day. On examination an abscess was found in the parietes of the left ventricle, which communicated both with the pericardium and the interior of the ventricle. In Mr. Howitt's case, the patient was eight years of age, and after complaining of an apparently neuralgic pain in the calf of the leg, became dull, and eventually comatose. On examination, an abscess was found in the walls of the heart which had opened into the pericardium, and right auricle. The pericardium contained a pint of purulent matter mixed with flakes of a cheesy consistence.

The *causes* of myocarditis are the same as of pericarditis, or endocarditis; and among these rheumatic fever, and prolonged exposure to cold after violent muscular exertion appear to be the most frequent. Myocarditis has been assumed by some writers to be a frequent cause of softening of the heart; and this softening, then, it is supposed gives rise to dilatation of the ventricles. But, dilatation is a very common occurrence, while myocarditis is an extremely rare one; besides, dilatation is most frequent at the

\* The Lancet, 1846.

† Ibid.

right side of the heart, which is scarcely ever the seat of myocarditis, and seldom even of endocarditis; and in a great many cases, dilatation is not associated with the results of inflammation either in the investing or lining membrane of the heart. According to Rokitansky, partial myocarditis when combined with endocarditis is a frequent cause of the acute form of aneurism of the heart described by him.

The *prognosis* in acute suppurative myocarditis, it is scarcely necessary to say, is very unfavourable, the disease runs a rapid course, and the best directed treatment fails; indeed every case hitherto recorded has had a fatal termination.

From what precedes we may conclude:

1. That acute myocarditis, uncomplicated with inflammation of any other part of the heart is very rarely met with.

2. That it is usually associated with inflammation either of the investing or lining membrane of the heart or with both; and, often in addition with pleuritis or pneumonia of the same side.

3. That it is scarcely ever general, being limited to the internal or external layers of the muscular fibres of the left ventricle; or being still more partial, and limited to a portion only of the walls of the ventricle.

4. That there are no symptoms which can be said to be pathognomonic of myocarditis.

5. That when pericarditis or endocarditis sets in suddenly, with intense violence, and runs a very rapid course, uninfluenced by remedies, there is presumptive evidence that the inflammation has extended to the muscular fibres of the heart in contact with the inflamed membrane.

6. That acute myocarditis may terminate in purulent infiltration, or in abscess of the walls of the left ventricle, and that either may occur without any prominent symptom referred to the heart being present.

## CHAPTER XVII.

DILATATION.—FORMS OF.—DILATATION WITH HYPERTROPHY.—PHYSICAL SIGNS.—SYMPTOMS OF.—DILATATION WITHOUT HYPERTROPHY.—PHYSICAL SIGNS, SYMPTOMS OF.—CAUSES OF DILATATION.—EFFECTS OF.—TREATMENT OF DILATATION.

LARGEMENT of the heart to any extent cannot occur without increase in the dimensions of its chambers, and the muscular fibres of the latter may under such circumstances preserve their normal thickness, be hypertrophied, or attenuated. The abnormality which really constitutes *disease* is dilatation; hypertrophy in present is to be regarded rather as nature's effort to enable the heart to maintain the circulation; for this reason, "simple hypertrophy" can scarcely be said to constitute a state of disease, "concentric hypertrophy," except as a congenital malformation, seldom met with. I propose therefore to consider morbid enlargements of the heart as varieties of *dilatation* rather than of hypertrophy.

Two principal forms of dilatation are met with:

1. *Dilatation with hypertrophy*, the active aneurism of the heart of Corvisart, the eccentric hypertrophy, hypertrophy with dilatation, or active dilatation of other writers.
2. *Dilatation without hypertrophy*, which will include the simple dilatation of authors; and dilatation with attenuation, the passive dilatation, or passive aneurism of the heart of the older writers.

## DILATATION WITH HYPERTROPHY.

This form of dilatation is met with in various degrees, sometimes the increase in thickness of the parietes is inconsiderable, sometimes it much preponderates over the dilatation. Both ventricles and auricles may be simultaneously affected, or it may be confined to one side of the heart, or to a single cavity, and the ven-

tricles are seldom engaged without their corresponding auricle suffering. The heart may preserve its normal shape, or this may be variously altered as dilatation or hypertrophy preponderates, or as one or more of its cavities are engaged.

Three varieties of dilatation with hypertrophy may be distinguished :

1. *Both ventricles and auricles* may be dilated and hypertrophied, when the heart attains the largest size it is capable of, being sometimes twice or three times larger than natural ; and its weight, which in the normal state is about nine ounces, may reach to forty ounces, in a few instances it has even exceeded this. Dr. Croker King communicated a case\* to the Surgical Society of Ireland, where the heart weighed forty-four and a half ounces, the enlargement being principally in the left ventricle. The largest heart I have ever seen is one preserved in the museum of St. George's Hospital, London, which is stated to have weighed five pounds. The shape of the heart varies as dilatation or hypertrophy is in excess ; when hypertrophy of the ventricles predominates, the organ preserves more or less its normal shape ; when dilatation predominates, it acquires a globular form. When the heart attains a large size, its position is more or less altered, it lies more transversely in the chest, and by its weight may help to depress the diaphragm, and cause the impulse to be felt on a lower plane than natural.

2. *The left ventricle alone* may be dilated and hypertrophied, or the disease may greatly preponderate in it ; the right ventricle then looks like an appendage to the left, and the septum is more convex towards the chamber of the right ventricle, and encroaches upon it. The diameter of the heart from above downwards is increased, and the apex extends lower and more to the left side than natural. The parietes of the left ventricle, under these circumstances, may have a thickness of from an inch and a-half to two inches, and its cavity may be so much dilated as to hold the close hand of the subject. Dilatation with hypertrophy of the left ventricle is accompanied by dilatation of the corresponding auricle, and very generally by some degree of the same change in the right cavities.

3. *The right ventricle alone* may be dilated and hypertrophied, or the disease may greatly preponderate in it, when the transverse diameter of the heart will be increased, the right ventricle will

\* Dublin Medical Press, vol. xiv.



extend below the left to form the apex of the organ, and the heart will exceed the præcordial limits on the right side, pulsating below the xyphoid cartilage, or to the right of this point. Dilatation with hypertrophy of the right ventricle is always accompanied by dilatation of the corresponding auricle, and generally by some change in the left cavities.

The increase in thickness of the parietes of the affected ventricles is not necessarily uniform throughout; it may preponderate in one part while others preserve their normal thickness, or may even be attenuated. The anterior and posterior walls of the ventricles are more susceptible of hypertrophy than the septum, and the columnæ carneæ are very generally increased in thickness when the other parts of the ventricle suffer.

Hypertrophy is not necessarily accompanied by any *morbid alteration* of the muscular tissue of the heart, though it often undergoes degeneration subsequently; the tissue is somewhat more dense, its consistence is greater, and its colour deeper than natural. "The consistence of the *right* ventricle presents (Rokitansky observes\*) a striking anomaly in the more highly developed forms of hypertrophy, the texture acquiring a toughness which is never observed under any condition in the left ventricle. The walls, which become rigid and retract on being cut, exhibit extreme resistance and hardness, and yield when struck a sound which, according to Laennec, resembles the tone emitted from hard leather. A similar relation is observed in active dilatation of the auricles when excessively hypertrophied."

It has been a question with pathologists whether the muscular fibres are increased in number or in volume, in this morbid condition. Dr. Blakiston† says: "When the natural colour was removed in the hypertrophied walls of the ventricle, he was unable to detect any difference in the minute structure of the muscular fibre from that of the walls of a perfectly healthy heart: measured with the aid of Powell's micrometer eye-piece, under a magnifying power of 500 diameters, the average breadth of the fasciculi was about 1,400th of an inch in both cases. Vogel, either from his own researches or those of Henle, has arrived at the same conclusion, and states that 'the volume of muscle is increased without the single fasciculi gaining in thickness; hence, it must follow

\* Pathological Anatomy, vol. iv.

† Diseases of the Chest.

that their number is increased, and that new ones have amongst those previously existing.' ”

#### SYMPTOMS OF DILATATION WITH HYPERTROPHY.

The physical signs and the symptoms of dilatation with hypertrophy vary according to the part or parts of the heart to the degree the disease has reached, and to the complications.

#### PHYSICAL SIGNS.

*Inspection.*—The heart cannot be dilated and hypertrophied without being increased in size, and it cannot be increased without exceeding the præcordial limits, and encroaching on neighbouring organs; its apex then instead of beating in its normal situation will be felt below this point, and more to the right side according to circumstances. When hypertrophy predominates and has lasted long, a bulging of the præcordial region is occasionally observed, more particularly in young subjects. The chest on inspection is unsymmetrical, projecting more on the left side, which is not only evident to the eye, but can be determined by measurement, though the difference is not as would be supposed from looking at the chest, seldom more than half an inch. This bulging of the præcordial region is, as remarked by Dr. Hope, a sign of only secondary importance, as it does not exist till the hypertrophy is very considerable, and it is sufficiently indicated by other signs. Besides, the chest is always naturally symmetrical. Dr. Swett\* says: “if we examine the chests of a large number of individuals we are surprised to find how few are perfectly symmetrical, and frequently there will be noticed a slight bulging or depression entirely in the parietes; one of the most frequent sites of bulging is the præcordial region.” On the other hand, in hypertrophy may be extreme, and this sign be absent. In the case which I have alluded to where the heart weighed forty-four ounces, instead of any bulging in the præcordial region there was absolutely flattened. In this instance, however, it was observed, the pericardium was adherent.

*Palpation.*—As a general rule the impulse is strong and natural, and usually in proportion to the increase in the

\* On Diseases of the Chest.

the parietes of the ventricles. In addition to being stronger, it presents the double character in a more marked degree, the diastole being accompanied by a very perceptible impulse. It is also felt over an abnormal extent of surface, and the systolic portion is gradual, heaving, and prolonged, elevating the head resting on the stethoscope. If the amount of hypertrophy be inconsiderable, the impulse will be but slightly increased, if considerable it will be strong and heaving in proportion, elevating the whole side of the chest, and followed by the "abrupt back-stroke" of Hope (the diastolic impulse). In extreme cases, "the whole person and even the bed is shaken by each contraction of the heart; in others the sensation is that of a large mass of flesh rolling or irregularly revolving in the chest." It sometimes happens, that in the very advanced stage, although a great amount of hypertrophy exists, the impulse is feeble and the heart's action irregular; this generally depends upon the circulation through the heart or lungs, or both being impeded; or it may be due to fatty or other degeneration of the muscular tissue, or to valvular complication.

*Percussion* is of little assistance to the diagnosis in the very early stage, and where the increase in size of the heart is inconsiderable; in the advanced stage however, it is capable of affording valuable aid. Thus when the heart is much enlarged, the lungs will be pushed aside, and a larger surface of the organ coming in contact with the parietes of the chest, a dull sound will be elicited by percussion over a wider area than natural; the degree of dullness and the resistance to the finger will also be increased. When the left ventricle is engaged, the site of the dull sound will be towards the left of the præcordial region, and low down; when the right ventricle is engaged, the site of the dullness will be towards the inferior portion of the sternum, and when both ventricles are dilated and hypertrophied, it will be perceived in both these situations. In extreme cases the extent of surface which yields a dull sound becomes very considerable; in that recorded by Dr. C. King the dullness on percussion is stated to have extended from the second left rib to the ninth, and from a line with the centre of the axilla to the right of the sternum, "the area of dullness being thirty-six square inches."

*Auscultation*.—In dilatation with hypertrophy the frequency of the heart's contractions and its rhythm are unchanged, but the

tone of the sounds, their relative duration, and the limits within which they are audible are more or less altered according to the amount of disease present, and according to its seat, whether upon the right or left side of the heart.

The general effect of hypertrophy upon the ventricles is to render the *first* sound of the heart duller than natural; at the same time, the systole being more slowly performed, the first sound is likewise prolonged. The *second* sound is not much altered unless the hypertrophy is in excess, when it becomes more feeble; and the interval of repose, owing to the prolongation of the first sound, is shorter than natural. The sounds are generally audible beyond their normal limits, but this is a sign of only secondary importance. In the very advanced stage, both sounds become feeble and indistinct.

When the right ventricle alone was dilated and hypertrophied, and sometimes when the left alone was engaged, I have on several occasions heard a *triple sound*, the reduplication being in the first sound, this was sometimes intermittent, sometimes persistent up to a short period before death. When the left ventricle is the principal seat of the disease, a bruit de soufflet accompanying the first sound is occasionally audible, particularly during attacks of palpitation, which disappears as the latter subsides: when persistent it is due to a valvular complication, more rarely to anæmia.

#### LOCAL AND GENERAL SYMPTOMS.

*Palpitation.*—When dilatation with hypertrophy is uncomplicated, when not in excess, and when the circulation is unimpeded, the subject of it generally makes little or no complaint of palpitation, although the impulse may be much increased, and felt over a comparatively large surface; he cannot however walk quickly, or ascend a stairs without suffering inconvenience. When the disease is advanced, palpitation in a greater or less degree is constantly felt, there are remissions but not perfect intermissions, and the patient at times suffers considerable distress from it. For an obvious reason, it is a more prominent symptom when the chest is narrow or deformed, or when anæmia is present.

*Dyspnœa.*—*Cough.*—In connection with palpitation, the patient is generally short-winded, and some dyspnœa is complained of, which in advanced cases may become a distressing symptom. If the

yspnœa amounts to orthopnœa, the disease is generally complicated with bronchitis, valvular disease, œdema or congestion of the lungs. In the latter cases cough is often a troublesome symptom, may be dry and hard, or soft and accompanied by copious expectoration. Hæmoptysis is rare, and seldom or never occurs in the uncomplicated form of the disease.

*Pulse.*—Dilatation with hypertrophy of the right ventricle exercises no influence upon the radial pulse, but when the left is engaged, and no valvular complication exists, it presents well-marked characters, being full and strong, “dwelling long under the finger,” and regular as long as the circulation continues free. The pulse also is said not to be so easily altered by changes in the posture of the patient as in the healthy subject; a remark first made by the late Dr. Graves. In extreme cases, and when complicated with obstructive valvular disease, or with degeneration of the muscular fibre of the left ventricle, it becomes feeble, intermittent, or irregular.

Dilatation with hypertrophy of the right may in general be distinguished from the same morbid state of the left ventricle, by the impulse in the former wanting the heaving character, and the latter wanting the firmness and strength which it has in the latter; the dulness on percussion extending more to the right side, and the apex beat being felt below the sternum, and to the right of the bone. In addition, dyspnœa and a tendency to hæmoptysis are more common in the former, while jugular pulsation in addition will be present when the right auriculo-ventricular orifice is dilated, and free regurgitation is permitted. When the left ventricle is principally engaged, the increased heaving, and double impulse, the apex beat being low down and to the left side, in advanced cases on a line with the centre of the axilla, will always enable us to diagnose it. Among these signs, the character of the impulse, and the situation in which the apex beat is felt are the most characteristic.

#### DILATATION WITHOUT HYPERTROPHY.

Two principal forms of dilatation without hypertrophy are distinguished, which differ as the parietes of the affected chamber or chambers are attenuated, or the contrary, viz :

1. Dilatation without attenuation, the simple dilatation of the ventricles.

2. Dilatation with attenuation, the passive dilatation of writers.

In *Dilatation without attenuation*, the capacity of the chamber is augmented while its parietes preserve their normal thickness; it constitutes the "simple dilatation of the heart" of the majority of writers. The latter however is not strictly speaking a correct term, a hollow organ cannot be distended beyond its normal limits without the parietes becoming thin in proportion; but in this instance, the parietes of the chamber are not thinner than natural, there must consequently be an addition to the muscular fibres, and what is termed simple dilatation is in fact a combination of dilatation with some hypertrophy.

In *Dilatation with attenuation* while the capacity of the chamber is augmented, its parietes are attenuated. It constitutes the passive aneurism of the heart of Corvisart, and was formerly termed "passive dilatation" in contradistinction to dilatation with hypertrophy, the "active dilatation" of writers. If the term attenuation is understood as synonymous with atrophy, and the muscular parietes are supposed to be atrophied in this morbid condition, the term, Dr. Latham\* remarks, is a misnomer. "Here there has been a lengthening and expansion of the muscular fibres, but no diminution of their substance. In obedience to a pressure from within they have yielded and spread themselves over a larger space. They have lost nothing. They have suffered no atrophy, only they have not, as in what is called simple dilatation, experienced a proportionate hypertrophy."

It is rare to find all the chambers of the heart dilated in the same subject, the disease is usually limited to one side of the organ, and the auricles having naturally thinner parietes more readily become dilated than the ventricles; while the right ventricle, having thinner walls than the left, is more often dilated than it. The septum of the ventricles less frequently suffers from attenuation than the other parts. When the ventricle upon one side is dilated, its corresponding auricle is almost always likewise engaged; and when dilatation predominates in one cavity, hypertrophy may predominate in another. Dilatation with hypertrophy of the left cavities is usually followed by dilatation with or without attenuation of the right cavities.

The size which the chambers of the heart, particularly the

\* On Diseases of the Heart, vol. ii.

uricles, sometimes attain is remarkable; these cavities, which in the normal state hold little more than two ounces of fluid, have been found so much dilated as to hold twelve or thirteen ounces. Dr. Parry's posthumous work\* contains a case where the right auricle held nine ounces, and the left thirteen ounces of coagulated blood. Dr. Gairdner† mentions another where the left auricle was capable of containing a moderate sized cocoa-nut, while the right could have held a billiard ball; the walls of both were slightly hypertrophied, and the ventricles were normal. In extreme dilatation of the auricles, the endo and pericardium, in the interspaces between the bundles of muscular fibres are in contact, and alone form the parietes at these points.

The shape of the heart is always altered in this form of dilatation, its transverse diameter is increased, its apex is rounded off, sometimes scarcely distinguishable, and the whole organ assumes more or less globular form.

Dilatation of the chambers is usually accompanied by some dilatation of the orifices of the heart; this is much more common at the auriculo-ventricular, than the arterial orifices; and, at the right, than the left side of the heart. According to Hasse‡ dilatation without attenuation is "associated with dilatation of the arterial orifices, and frequently with preternatural width of the whole course of the aorta and pulmonary arteries;" while dilatation with attenuation is "concurrent with enlargement of the auriculo-ventricular orifices, and of the veins opening into the heart." When the orifices are dilated, the curtains of the valve increase in size, and their tendinous cords, and fleshy columns come lengthened, by which the valves are rendered competent, and regurgitation is prevented. If the valve remains of the normal size, it will be incapable of closing the orifice, and regurgitation will be permitted. This, which is very frequent at the tricuspid orifice, is comparatively rare at the mitral.

Dilatation is not necessarily accompanied by any morbid alteration of the muscular tissue of the heart. When it has lasted long, however, and has gone on to a fatal termination, we generally find its colour to be deeper, and its tissue softer, more flabby, and more easily torn than natural. In general but little fat is

\* Collections from unpublished writings. † Path. Anat. translated by Swaine.

‡ Brit. and For. Med. Chir. Rev. vol. xii.



found upon the surface of the heart in this diseased state, but partial fatty degeneration of the muscular fibre is by no means uncommon.

#### PHYSICAL SIGNS.

*Inspection.—Palpation.*—Increased thickening or attenuation of the parietes of the ventricles may be pretty accurately estimated by the strength and other characters of the impulse. The effect of hypertrophy when combined with dilatation of the ventricles we have seen to be to render the impulse stronger than natural; the effect of attenuation combined with dilatation is to diminish the strength of the impulse. Again the effect of hypertrophy is to render the impulse prolonged and heaving; the effect of dilatation is to render it short.

The strength and other characters of the impulse differ as the dilatation is, or is not accompanied by attenuation. In dilatation *without* attenuation, the impulse is abrupt, and circumscribed, it presents the double character in a more marked degree than the healthy heart; it is likewise felt beyond the normal limits, and more to the right or left side, as it predominates in the right, or left ventricle. In dilatation *with* attenuation, the impulse is less marked, and less strong than that of the healthy heart, it is also shorter, often unequal, and irregular. When the dilatation is accompanied by softening, the impulse may be altogether absent, the ventricular systole being too feeble, and the shock too slight to be communicated to the parietes of the thorax.

In dilatation of the right side of the heart, with or without attenuation, a distended condition of the jugular veins is generally present, seldom when the left side of the heart alone is engaged. The distension at first is only temporary, eventually it becomes permanent; it is observed upon both sides of the neck, though sometimes predominating upon the right. Its cause is simply mechanical, the venous current in the superior cava being impeded, its branches become distended. When the tricuspid orifice permits free regurgitation, and the systole of the right ventricle is sufficiently strong to communicate a backward impulse to the blood descending by the superior cava, a pulsation in addition is observed in the jugular veins at the root of the neck, sometimes upon both sides, occasionally limited to the right side. This does



not occur in dilatation with attenuation, or softening of the right ventricle, the systole being too feeble to communicate a backward impulse to the venous current.

*Percussion.*—In dilatation with and without attenuation the transverse diameter of the heart is increased, the lungs are encroached upon, and a larger portion than natural of the surface of the heart comes in contact with the parietes of the chest, hence a dull sound is elicited by percussion over a wider area than natural, and as the dilatation predominates upon the right or left side, it will be most marked over the lower portion of the sternum, or to the left of this bone. Dilatation with and without attenuation may it is said be distinguished from dilatation with increased thickness of the parietes by the degree of resistance to the fingers employed in percussion being less in the former than the latter. Or, if the amount of blood which distends the cavities is diminished by bleeding, the area of præcordial dulness will, it is supposed, be diminished in the former, but not in the latter. These rules, although they may look very well upon paper, are not to be depended on, and percussion alone would never enable us to distinguish dilatation *with* from dilatation *without* increase in thickness of the parietes.

*Auscultation.*—The first sound of the heart in its healthy state is prolonged compared with the second, and in dilatation with hypertrophy of the ventricles it is still more prolonged; while in dilatation without hypertrophy it becomes shorter than natural, resembling the second sound, so much so as to render it difficult, if the heart's action is very rapid, to distinguish the first from the second sound.

The general effect of dilatation, we have seen, is to impair the muscular power of the ventricles, to weaken the systole, and to diminish the ability of the heart to maintain the circulation. This cannot occur without exercising some influence upon the heart's sounds, which become more feeble than natural, while the action of the organ is more rapid than in health. When dilatation is in excess, or when it is combined with softening or degeneration of the muscular fibre, the sounds are not only feeble, but irregular.

Clearness and loudness of the sounds of the heart were laid down by Laennec as signs of dilatation of the ventricles; but, the cases in which the sounds present these characters are rather ex-

amples of functional derangement of the heart, where the organ contracts with energy, and a spasmic condition of the blood perhaps likewise exists.

In dilatation predominating in one ventricle, a *triple sound* is occasionally audible, and it is the first sound which is then usually doubled; I have more frequently met with it in dilatation *with* than without hypertrophy, and where the right ventricle was engaged than the left. When the auriculo-ventricular orifice is dilated, and its valve is incapable of closing the orifice, a murmur from regurgitation may accompany the first sound of the heart. Although regurgitation from this cause is very frequent at the tricuspid orifice, a murmur is very rare. This condition of the orifice and valve is uncommon at the mitral orifice; when it does occur a murmur will accompany the first sound, provided the parietes of the ventricle are not softened, or attenuated.

*Dilatation of the auricles.*—There are few physical signs peculiar to dilatation of the auricles; as a general rule, almost, when a ventricle is dilated its corresponding auricle participates in the dilatation; in cases of considerable contraction of the mitral orifice, however, where the left ventricle is not only not dilated, but where its cavity is more often actually diminished in size, the left auricle is always dilated, and its parietes are generally likewise hypertrophied.

According to M. Pigeaux, extreme dilatation of the auricles may be detected by the dull sound yielded by the parietes of the thorax over their site, and by the diminution or absence of the respiratory murmur at the same part of the chest, provided no pulmonary lesion is present which could occasion either dulness on percussion, or absence of the respiratory murmur. When the right auricle is much dilated, the large veins of the neck are, in addition, permanently dilated. When the left auricle is much dilated, and its parietes are hypertrophied, “a faint pulsation or undulation between the second and third left ribs” (first noticed by Dr. Blakiston,) is occasionally present; while the long continued pressure of the auricle upon the left bronchus may cause flattening of this tube, a sign to which attention was first called by Mr. W. King.

Post-mortem distension of the auricles must not be confounded with dilatation of their chambers. The former is usually met with

es where death has been gradual, and the pulmonary circulation had been much impeded previously. The distension of the is generally nearly uniform throughout, while, in cases of ion, there is often hypertrophy of some portion of the parietes.

#### LOCAL AND CONSTITUTIONAL SYMPTOMS.

he local and constitutional symptoms of this morbid state according to the part of the heart principally engaged, and ge, according as pulmonary congestion and imperfect aëration a blood are present, or as the arterial circulation is enfeebled he systemic capillaries are congested: according to its com- ion with valvular disease, or softening of the muscular tissue ; heart, or with morbid states of the bronchial tubes, lungs, or kidneys; and, as anæmia, dyspepsia, the gouty diathesis, re present.

*palpitation.*—The effect of dilatation when combined with ation being to weaken the muscular power of the ventricles, o increase the difficulty of expelling their contents, more or alpitation is constantly present, while it is aggravated by cir- ances which would have no effect upon a healthy heart, such s of coughing, mental emotion, or muscular exertion. On ing the hand or the stethoscope to the præcordial region, igh the patient's sensation of palpitation is strong, the impulse aker than natural, or the heart's action is oppressed and ring. This is also observed in dilatation without attenuation, muscular tissue of the heart is softened. In advanced stages e disease when the difficulty in transmitting the blood in- a, the patient suffers from distressing sensations of oppression, ig, or anginal pain, and the heart's action may suddenly from inability to expel its contents; or fibrinous concretions orm in the chambers of the heart, which obstruct the orifices, npede the action of the valves, and cause death as certainly, h more gradually.

*dyspnœa.*—*Orthopnœa.*—When dilatation is not advanced, uncomplicated, and when the circulation through the heart mpeded, dyspnœa is not a prominent symptom, it is rather a ion of oppression, or “smothering,” which is experienced ularly on ascending stairs, or on making some unusual exer-

When the chambers of the heart are much dilated, however,

whether the parietes are hypertrophied or not and it has led to congestion of the pulmonary capillaries, or the patient has suffered frequent attacks of bronchitis, dyspnœa passing into orthopnœa becomes a very prominent symptom; it occurs in paroxysms of extreme severity, the patient suffers intense distress in them which is depicted in his countenance; the respiration is gasping, or wheezing, accompanied by cough, and expectoration of a sero-mucous liquid, sometimes streaked with blood, or of a more viscid mucus which may be deeply coloured with blood. Until within the last few days or weeks of life, the dyspnœa in those cases is not constant, the patient may be comparatively free from it for many hours at a time, but it seldom fails to come on at night, and generally soon after he falls asleep, obliging him to rise and pass great part of the night in a chair, or to sit up in bed propped up by pillows.

*Pulse.*—No information is to be derived from the pulse when the dilatation is limited to the right ventricle, or to the auricles; when the left ventricle is much dilated, or when the dilatation is combined with flabbiness or softening of its muscular tissue, the pulse is soft, feeble, or small, and habitually intermittent or irregular, particularly so during paroxysms of dyspnœa. In general it is quickened, though Mr. Burns\* regarded slowness of the pulse as a sign of dilatation; he says that in the cases of genuine unmixed dilatation met with by him, the pulse was uniformly slower than natural, sometimes remarkably so; and he relates a case where the pulse, which in health had been seventy or eighty, came down to eighteen, and eventually as low as ten or eleven.

*Passive hæmorrhage.*—Passive hæmorrhage from some portion of the mucous surface is not uncommon in the advanced stage of dilatation; it is seldom however profuse, and the blood escapes by exudation, not by rupture of vessels. When the pulmonary veins have much difficulty in unloading themselves, and the pulmonary capillaries become congested, the blood may escape upon the surface of the broncho-pulmonary mucous membrane, and the expectoration becomes mixed with blood. The sputa, in such cases, are either viscid, expectorated singly, and of a very dark colour, or they are less viscid, and have a brighter colour. In some cases hæmoptysis to a considerable amount occurs, and the lungs after

\* Treat. on Diseases of the Heart.

ath exhibit the appearances familiar to us as pulmonary apoplexy.

When the venous system throughout the body has become congested, nature sometimes endeavours to relieve it by allowing blood to exude upon the mucous surface of some part of the alimentary canal, generally the stomach, or small intestines; or, by permitting its escape from the hæmorrhoidal vessels. "When the hæmorrhage proceeds from the mucous membrane of the stomach, and the blood is not immediately ejected, it may have (as Dr. Keene remarks) the appearance of coffee-grounds, in consequence of being exposed to the action of the gastric juice; in the intestines it is often blackened by the intestinal acids." "It is common, under these circumstances (he adds), to find the mucous membrane after death so vascular as to present the appearance of inflammation. This is especially the case in the stomach and intestines; and it is necessary to be aware of it in order to guard against the error of attributing the redness to inflammation."

*Congestion of the Liver and Kidneys.*—After the lungs, the liver is the organ next most frequently the seat of congestion in cases of dilatation. The hepatic and portal circulation both being impeded, the amount of blood retained in the organ is augmented, and its size is sensibly increased, indicated by percussion; while slight jaundice is occasionally superadded. The kidneys likewise when become congested in the advanced stage of dilatation, they become enlarged, pain in the lumbar region is experienced on strong pressure; and, according to some authorities there is increased dulness on percussion over their site. The urinary secretion is scanty, high-coloured, usually loaded with lithates, and sometimes albuminous.

*Cerebral Symptoms.*—The circulation in the brain is sometimes impeded in these cases, and more or less congestion of this organ may be the result, evidenced by headache, referred sometimes to the site of the longitudinal sinus, by heaviness, somnolency, tinnitus aurium, muscæ volitantes, injection of the conjunctiva, &c. When softening of the muscular tissue of the heart is combined with dilatation, attacks resembling syncope are common, apparently owing to an insufficient supply of blood being transmitted to the brain.

*Dropsy.*—Few cases of dilatation of the ventricles run their

course without the supervention of dropsy, which always commences as anasarca about the ankles, extends to the face, trunk, and upper extremities, usually being more marked in one hand or arm, and in one side of the face. As in the dropsy of obstructive mitral valve disease, the lower extremities, the scrotum and penis in the male, and the labiæ in the female, sometimes attain an enormous size, by which the patient's sufferings are greatly aggravated.

The exciting cause of the dropsy in both instances is the same, the pulmonary and general venous circulation being impeded, the systemic capillaries relieve themselves by allowing the watery portion of the blood to transude. In both instances, likewise, tricuspid regurgitation generally precedes dropsy, and it is to it, rather than to the valvular disease, or dilatation, that Dr. Blakiston\* refers the obstruction which leads to anasarca. "When regurgitation to any extent takes place, it is impossible (he says) to conceive a more powerful obstruction than is then offered to the passage of the blood from the veins into the heart, by a strong counter-current forced backwards by the systole of the right ventricle.

*Countenance.*—A dusky or purple hue of the face, particularly of the cheeks and lips, was a symptom of dilatation of the right chambers of the heart upon which Corvisart laid much stress. Laennec regarded it rather as a sign of dilatation with hypertrophy of the same part. It is a common symptom in the advanced stage of dilatation of the right side of the heart, but it is not observed until pulmonary congestion, and deficient aëration of the blood have supervened, and it is most frequent where the dilatation is accompanied by bronchitis, and emphysema of the lungs, or has followed obstructive valvular disease at the left side of the heart. In dilatation with attenuation of the left ventricle, the countenance is often pale, bloated and anasarcous, the temperature of the surface is lower than natural, and the feet and hands are cold and blue.

#### CAUSES OF DILATATION.

Dilatation, whether accompanied by much or little thickening of the parietes of the cavity engaged, is almost always a *secondary*

\* On Diseases of the Chest.

fection; and the circumstances under which it occurs are sufficiently numerous; some of these tend more immediately to induce hypertrophy, some dilatation; while others are liable to be followed by both these states. Thus, obstructive lesions of the valves & orifices as a general rule, give rise to hypertrophy of the parietes of the chamber immediately behind them in the course of the circulation; regurgitant lesions to a predominance of dilatation in the chamber into which the blood regurgitates. The same mechanical impediment may, however, give rise to excess of hypertrophy in one subject, and to excess of dilatation in another. For instance, if it is slowly and gradually developed, if the patient's constitution is unimpaired, his general health good, and the tissue of the heart sound, hypertrophy will most probably preponderate. Whereas, if the mechanical impediment is suddenly or rapidly induced, if the patient's constitution is broken down, or the tissue of the heart weak, dilatation will be more likely to preponderate.

It is a common opinion that dilatation is *always* the result of some mechanical impediment to the circulation, and the cases are regarded as exceptional in which no valvular lesion is found after death. This however is by no means a correct view, the examples are sufficiently common in which none can be discovered; and it is a remarkable fact, that, in the most extreme instances on record of dilatation with hypertrophy, and where the heart attained the largest size, no valvular lesion of any kind existed.

The accompanying table from Dr. T. K. Chambers' *Decennium Pathologicum*,\* shows the frequency of dilatation or hypertrophy in 505 cases, and the number of times they were associated with valvular disease, or with disease of the aorta:

FORM OF CARDIAC DISEASE.	Valves chronically diseased.	Aorta diseased— valves healthy.	Both healthy.	Total.
Dilatation . . . . .	87	53	69	209
Hypertrophy and Dilatation . . .	89	15	35	139
Hypertrophy . . . . .	74	23	40	137
"Enlargement" (form not specified) .	4	3	13	20
Total . . . . .	254	94	157	505

The following are Dr. Chambers' conclusions under this head:

\* British and Foreign Med. Chir. Review, vol. xii 1853.



“As a general rule, the most common diseased condition of the muscular walls is dilatation.

“With diseased valves, dilatation with or without corresponding hypertrophy, is about equally common.

“With diseased aorta, dilatation alone is most common.

“Where both valves and aorta are healthy, simple dilatation is *by far* the most common.”

It would appear as if a constitutional predisposition to dilatation existed in some subjects; the parietes of the heart from birth being thinner, or its tissue more lax than natural. Again, young persons, though with originally healthy hearts, if called upon to make unusual or protracted exertions, before the muscular system is fully developed, and if they are at the same time badly nourished, are liable to become the subjects of dilatation.

Habits of intemperance, particularly when contracted early in life, seem to be a fruitful source of dilatation, with or without hypertrophy. It would appear to act at first by stimulating the heart to increased action; when it has made inroads upon the constitution, and the liver, kidneys, &c., have become congested, the free passage of the blood through these organs being impeded, this reacts upon the left side of the heart, and upon the ascending portion of the arch of the aorta, which becomes dilated, while its interior at the same time often becomes rough from adventitious deposit. Eventually, owing to the morbid state of the blood, the tissue of the heart often becomes flabby and softened.

Frequent and prolonged muscular exertions in healthy or plethoric individuals, are by no means unfrequent causes of dilatation with a predominance of hypertrophy of the left ventricle. The occurrence of hypertrophy under such circumstances was explained by Corvisart, by the coronary arteries conveying an increased amount of blood to the tissue of the heart, owing to these vessels being the first given off; increased afflux of blood leading to increase of nutrition, and increased nutrition to increase of volume. In addition, the coronary circulation takes a shorter time to be completed than the general circulation; in other words, the blood conveyed by the coronary arteries is returned by the coronary veins more rapidly than that transmitted to the general system; hence, when the heart's action is quickened, this organ receives more blood in proportion; and if the causes continue long enough



in operation, hypertrophy is liable to follow. In ill-fed and anæmic individuals, on the other hand, where the blood is impoverished, or the patient debilitated by previous illness, increased action of the heart will be followed rather by dilatation of the chambers, the weak and flaccid tissue of the organ yielding to the distensile force of the blood.

In the majority of cases, the exciting cause of dilatation is an impediment or mechanical obstruction to the passage of the blood out of the chambers of the heart. The parietes yield to the distending force from within, and the cavities become enlarged; when this has persisted for a period which varies in different cases, the stretched muscular fibres lose their elasticity, the cavity is incapable of returning to its ordinary dimensions, and becomes permanently dilated. As a general rule, it "depends upon the proportion which the resistance of the muscle bears to the distending force," whether the dilatation be accompanied by increased thickness of the parietes of the cavity engaged, or not; in the unhealthy subject dilatation will preponderate, in the healthy subject hypertrophy will preponderate. And Dr. Chevers has shown that in a heart in which hypertrophy *had* been the prevailing lesion, dilatation may become the prevailing lesion, "under the combined influence of prolonged and increasing obstruction, congestion, and failing health."

Corvisart compared the mode in which dilatation of the chambers of the heart succeeds their frequent distension, to the effects of retention of urine upon the bladder. Burns looked upon dilatation of the heart and enlargement of the gravid uterus as analogous phenomena; "only in the one case the increase in size tends to a useful purpose, whereas in the other it is *ab origine* morbid." On the other hand, Mr. Greeves has advanced the opinion that dilatation is not the passive result of long-continued distension of the cavities; he supposes that the external spiral fibres of the ventricles antagonize the internal layer, and that the process by which dilatation is effected is an active rather than a passive one.

Dilatation is frequently, as I have said, the result of some impediment or mechanical obstacle to the current of the blood; but the degree of dilatation or hypertrophy is not necessarily in proportion to the amount of the impediment; "being (as Dr. Peacock

remarks) in some cases very great when the obstruction is trivial, and in others slight, when very great obstruction exists. In dilatation of the left side of the heart, the obstruction is generally seated upon the endocardial surface, and either at the valve or orifices; in dilatation of the right side, it often has its seat anterior to the heart, viz., in the lungs, or at the parietes of the chest. For instance, a contracted state of the aortic orifice, disease of its semilunar valves, a tumor compressing the aorta, an aneurismatic state of the interior of this vessel, or a diminution or loss of elasticity in its coats, may each give rise to this condition of the *left* ventricle; while any impediment to the free passage of the blood through the lungs, such as is produced by chronic bronchitis, dilated bronchial tubes, emphysema, asthma, or congestion of the pulmonary capillaries, may be followed by this morbid condition of the *right* ventricle. Under the latter head may be included malformation of the chest, whether congenital, or from disease of the dorsal spine in early life, and probably also trades or occupations requiring continued muscular exertion in a constrained posture.

Dr. Barlow\* has shown that "defective expansion of the lungs in early life," is no unfrequent cause of dilatation, with or without hypertrophy of the right side of the heart; this may be the result of previous disease of the lungs or pleura, or of congenital malformation with contraction of the chest, or of narrowing or deformation of this part from disease or injury. According to Dr. Gairdner the great majority of cases of hypertrophy with dilatation, independent on valvular disease, have their origin in chronic disease of the lung, accompanied by partial atrophy of its tissue, and usually also by emphysema; which, he has shown to be the almost invariable consequence of such atrophy.

There appears to be a close relation between albuminuria and dilatation of the heart, but whether as cause or effect pathologists are not agreed, though they generally incline to the former. Dr. Barclay† says, "some idea may be formed of the frequency of the association, from the circumstance that in going over the *post mortem* records at St. George's hospital for a period of about thirty years, he found 141 cases in which the kidney is noted as evidently

\* Gulstonian Lectures, Med. Gaz.

‡ Med. Chir. Trans vol. xxv.

† Med. Chir. Rev. vol. xii.

diseased; and among these hypertrophy is mentioned as occurring fifty-five times, and dilatation thirty-six times, either together or separately in sixty-three individuals; while there were only seventy-eight instances (very little more than half) in which the heart retained its natural dimensions." I am, however, rather inclined to regard albuminuria as an effect, than as a cause of dilatation; the great majority of the cases in which the association exists being examples of valvular unsoundness, upon which dilatation has supervened.

According to Dr. T. K. Chambers, *general obesity* is in itself a cause of dilatation. We cannot be surprised at this, he observes, "when we reflect on the great increase of work which is thrown on the chief organ of the circulation, by the increased area of capillaries through which the blood has to be propelled in fat people." "The addition of several stone to the weight in fat requires certainly a very large, though not perhaps a proportionate addition of blood and blood-vessels to nourish it, yet the same heart has still to undertake this extra labour. The balance, then, between the systemic and pulmonary circulation must be destroyed, and the lungs be unequal to the secretion of so much more carbon than they were made for; hence the blood becomes more venous, more liable to form congestions, and to dilate the cardiac cavity by its retarded pace."

Dilatation, whether with or without hypertrophy, is seldom limited to one chamber of the heart; although it originates in one cavity, or preponderates on one side, a minor degree of the same condition is usually observed on the opposite side of the heart; and this applies particularly to cases where the dilatation is independent of valvular disease.

#### EFFECTS OF DILATATION.

The effects of dilatation vary according to the part or parts of the heart engaged, according as dilatation or hypertrophy preponderates, according to the degree it has reached, according to the pathological state which produced it, and according to its complications. Its effects are manifested partly upon the heart, partly upon the current of the circulation, and partly upon the system at large; upon the heart, by leading to alterations in its size, shape, and position in the chest; upon the circulation, by retarding and

impeding the general venous current, and leading to insufficiency of the auriculo-ventricular valves; and upon the system generally, by inducing congestion of internal organs, and leading to serous effusion and hæmorrhage.

The alterations in *shape* which the heart undergoes, as dilatation or hypertrophy is in excess, or as one cavity only, or the entire organ is engaged, are sufficiently characteristic. Thus, when dilatation much preponderates, and when *both* ventricles are equally engaged, the heart acquires a *globular* shape, the transverse exceeds the vertical diameter, and the apex almost entirely disappears. When the *right* ventricle alone is considerably dilated and hypertrophied, it forms the apex of the heart instead of the left, this part is more obtuse than natural, and the transverse diameter of the heart is increased. When the *left* ventricle alone is considerably hypertrophied, the apex is lengthened and more pointed than natural, while the vertical diameter of the heart is increased. When the *left* ventricle is considerably dilated, while the right preserves its normal size, the latter looks like an appendage to the left, and the apex is more obtuse than natural.

The *position* of the heart, when this organ is considerably enlarged, becomes somewhat altered; it lies more transversely in the chest than natural, "the apex being directed to the left, and the base to the right side;" and the angle at which the veins on each side, particularly the left, enter the auricles, must be altered. This can scarcely occur, as Hasse remarks, without the return of the blood by these veins being impeded, and pulmonary or general venous congestion induced.

The effects of dilatation upon the circulation are much more prejudicial than of hypertrophy. The more dilatation is in excess, the weaker must necessarily be the propulsive power of the ventricles; and if the left is engaged, not only will it be incapable of propelling the arterial blood to remote parts, but the return of the venous current will be impeded or retarded. In addition, the chambers of the heart cannot become much dilated, without the orifices of communication between the auricles and ventricles suffering some change either in *size* or *shape*. This is observed much more frequently at the right than at the left side of the organ, and the tricuspid orifice often in consequence permits free regurgitation.

The effects of excess of dilatation upon the system at large are likewise much more prejudicial than of excess of hypertrophy; the tendency of the former is to give rise to venous congestion, and dropsy, the tendency of the latter is rather to produce arterial congestion, occasionally leading to hæmorrhage. Indeed, the ultimate effects of excess of dilatation are very nearly similar to those of advanced valvular disease, both having their origin in impeded circulation—impeded circulation through the heart being sooner or later followed by impeded pulmonary circulation, by congestion of the lungs, liver, &c., and by dropsy of the cellular tissue, and serous cavities.

Pulmonary and cerebral apoplexy are regarded by some pathologists as frequent effects of hypertrophy of the ventricles, in the former, of hypertrophy of the right, in the latter of hypertrophy of the left ventricle; the hypertrophied ventricle being supposed to propel the blood with increased force to the lungs or brain. The late Dr. Hope strongly advocated this view—speaking of the connexion between hypertrophy of the heart and apoplexy, he observes, “instances of apoplexy have been so frequently noticed that the relation of the two as cause and effect is, in my opinion, one of the best established doctrines of modern pathology. Eight or nine cases of suddenly fatal apoplexy, and numerous cases of paralysis from hypertrophy have within a few years fallen under my own observation. In the majority of these the patient exhibited what is commonly called the apoplectic constitution; that is a robust conformation, a plethoric habit, and a florid complexion. In others, these characters were absent; but the total number of the cases of apoplexy from hypertrophy is much greater than I have witnessed during the same period of apoplexy from causes independent of hypertrophy; whence, I am led to believe with Richerand, Bertin, and Bouillaud that hypertrophy forms a stronger predisposition to apoplexy than the apoplectic constitution itself.”\*

The influence of hypertrophy of the ventricles, in giving rise to pulmonary or cerebral apoplexy, appears to me to have been much overrated. In fact, hypertrophy is generally, as we have seen, a *secondary affection*—in the right ventricle the result of chronic bronchitis with emphysema of the lungs, or contraction or malformation of the chest, or more remotely of disease of the mitral

\* Treatise on Diseases of the Heart.

valve or orifice—in the left, of valvular disease at the aortic orifice, or of diseased states of the aorta, or of some other impediment to the free egress of the blood from the ventricle; in neither case would the blood be transmitted to the lungs or brain with augmented force, or in increased amount, but in each the return of the venous blood from these organs would probably be impeded; and, in the case of the brain, the actual amount of arterial blood transmitted to it might be so much diminished as to be insufficient for its due nutrition.

Moreover, hypertrophy in addition to being a *secondary* affection, is always (as one of the ablest writers on the subject observes) a *compensatory affection*. “After a very long and careful investigation of diseases of the heart, I have never (Dr. Chevers\* observes) seen hypertrophy unattended with more or less dilatation either of the cavity of the hypertrophic ventricle itself or of the artery proceeding from it. I have never been able to discover that the heart is susceptible of being rendered too muscular and too powerful from mere hypernutrition. In fact I have not seen or heard of any case in which it was demonstrable, that excessive muscular development and strengthening of the walls of the heart, existed otherwise than as a compensation for impediment of some kind, or for the weakening attendant upon temporary or permanent dilatation of its cavities. Nor have I been able to meet with a heart in which the amount of hypertrophy appeared to be more than *compensatory* for the coincident dilatation or impediment. Whereas, in nearly every complete case, the symptoms during life, as well as the sum of the morbid appearances, rendered it almost a matter of demonstration that the amount of hypertrophy was not, and had never been, fully sufficient to counteract the impediment or embarrassment to which the organ was subject.”

#### TREATMENT OF DILATATION.

In entering upon the treatment of dilatation, it must be borne in mind that it is not only a chronic affection, the growth of months or years, but that it is, in general, likewise, a secondary affection, and the causes which have called it into existence have been in operation for a lengthened period; while in the majority of cases the latter cannot be removed. In its early stage too, and

\* Treatise on Diseases of the Heart, Calcutta, 1851.

when moderate in degree it occasions so little inconvenience that we are seldom consulted at this period; while in its advanced stage it is little under the influence of remedies directed particularly to it; medical treatment being capable of doing little more than palliating, or removing its effects.

In considering dilatation with a view to treatment, and in taking a wide view of the cases of this disease, they seem to arrange themselves naturally under one of two general heads, though between the extremes there is every diversity of degree.

1. There is a dilated, but weak or flabby heart, the parietes of which may, or may not be hypertrophied; and the feeble organ is incapable of propelling the blood to remote parts, or perhaps of emptying itself completely.

2. There is a dilated and hypertrophied heart, capable of acting with vigour, and fully equal to performing its functions, but unable to do so owing to valvular obstruction, or some more remote impediment to the circulation.

In either case the indications of treatment are—

1. To endeavour to remove the cause of the dilatation if it be practicable.

2. To strengthen the parietes of the ventricles, to guard against over-distension of the chambers of the heart, and thus to stay the increase of the dilatation.

3. To relieve anæmia, debility, bronchitis, derangements of the digestive organs, and other complications of dilatation when present.

4. To diminish or remove venous congestion and the other effects of impeded circulation, without debilitating the patient.

5. To improve the general health, to support the patient's strength, and to maintain as tranquil a state of the circulation as possible.

When dilatation is secondary to valvular disease, or when it is followed some of the other morbid states which have been enumerated, the removal of the cause is beyond our reach. On the other hand when it arises in subjects debilitated by previous illness, when it comes on in chlorotic individuals, or in subjects weakened by loss of blood, when it has succeeded frequent attacks of bronchitis, or has apparently followed prolonged, or unusual muscular exertion; by directing our treatment to the cause, we can



do much for the patient's relief, provided the disease is not in a very advanced stage, and no serious complication exists, and provided the tissue of the heart is otherwise sound, and the patient young.

In order to stay the increase of dilatation, our treatment must be directed to preventing over-distension of the chambers of the heart, to giving tone to the organ, and to improving the general health. All measures likely to induce debility are to be avoided, hence, antiphlogistics and sedatives generally are contra-indicated. Among the latter, digitalis though a favourite remedy for quieting inordinate action is objectionable here; the palpitation and increased action of the heart in these cases are nature's means to enable the heart to maintain the circulation, and if we diminish its power of doing so by antiphlogistic treatment, or the administration of sedatives, we counteract the efforts of nature, and favour over-distension of the chambers. "Power (Dr. Chevers observes) is the great quality in which the diseased heart is wanting, and from first to last, our main indication of treatment is to husband that muscular energy which is gradually and inevitably declining even when its hypertrophy is greatest, and its contractile efforts are most tumultuous."

In order to diminish or remove venous congestion, and the other effects of impeded circulation, the same measures are to be employed as have been noticed under the head of valvular disease, viz., local abstraction of blood, cathartics, diuretics, expectorants, &c. But where dilatation is in excess, or the muscular tissue of the heart is softened, we should be very guarded in the employment of measures likely to increase debility; we must be content with means which will equalize the circulation, and determine to the surface; and as it is sometimes unsafe to abstract blood even locally, we must endeavour to produce the same effects indirectly by diminishing the amount of the fluid ingesta, by diaphoretic medicines, by rest, diet, &c. On the other hand, when the muscular tissue of the heart is neither flacid or softened, and the patient is not debilitated, hydragogue cathartics are often serviceable, by unloading the portal circulation, and the patient is generally sensible of the relief they afford. Indeed, when spontaneous diarrhæa occurs in such cases, it is remarkable how quickly pulmonary and hepatic congestion are sometimes relieved.



Although there are few more effectual means of relieving internal congestion than venæsection, we must be very guarded in employing it here; few cases in which dilatation predominates will bear it. Local bleeding, however, is not open to the same objection, and is often used with advantage, particularly when sudden aggravation of dyspnœa occurs, or a recent bronchitis is superadded to the other symptoms; here cupping between the scapulæ, or a few leeches to the præcordial region are often followed by immediate present relief to the dyspnœa, and palpitation. When the patient is considered to be too weak to bear loss of blood, dry cupping must be substituted for it, and repeated as occasion requires.

After local bleeding, or independent of it, there is no better means of relieving pulmonary and hepatic congestion, and of promoting the biliary and renal secretions than a mercurial course carried so far as to touch the gums; and the ordinary combination of calomel, or blue pill with squill, and digitalis, answers better than any other for this purpose; and in protracted cases, it may sometimes be had recourse to over and over again in the same subject, and each time with present relief.

When hypertrophy predominates setons or issues in the region of the heart have sometimes been found of use. M. Romberg considers that setons are best adapted to hypertrophy occurring in young subjects, and he cautions us against expecting too much from them at first; a persistence in their use for months or years is, he thinks, necessary, especially in cardiac disease of rheumatic origin; in some instances they appeared at first to aggravate the symptoms. The inconvenience and annoyance which setons or issues occasion are, however, almost sufficient to counterbalance any benefit they are capable of affording.

In order to maintain a tranquil state of the circulation, repose both of body and mind are indispensable, every thing capable of hurrying the circulation or exciting strong mental emotion is to be avoided; strict rules respecting diet must be laid down, and a healthy state of the secretions maintained. The food should be nourishing and easy of digestion, and but little fluid should be taken at meals; the amount of blood will thus be diminished, while its quality will not be deteriorated. Every kind of food likely to occasion flatulence is to be avoided, as nothing adds more to the

distress of the patient than flatulent distension of the colon. Medicines calculated to relieve the dyspeptic are generally called for, and the particular medicine or course must vary according to the peculiarities of each case.

Much of the success of treatment in cases of this kind depends upon the patient himself, and upon his observance of the rules of diet and exercise. If he lives intemperately, or is obliged to labour for long hours, or is exposed to all the vicissitudes of the weather, and the disease is advanced, there is little hope of benefitting him. But Burns\* observes, "he keeps quiet, submits to a regular digestible diet, and employs occasionally remedies to relieve dyspeptic symptoms, it will afford him comfort to know that he may ward off the fatal issue for a considerable time, and enjoy tolerable health."

When *Dropsy* supervenes, the prognosis becomes unfavourable, and the difficulty of affording relief becomes materially increased. "Of all forms of organic dropsy, the most dangerous depends on dilatation of the heart, with softening of the brain, and appears to be that (Dr. M'Dowell† observes) in which the effusion is least efficacious. We occasionally see even extreme dropsy, the effect of obstructive valvular disease, abate with great rapidity under judicious treatment. Not so, in dropsy which depends on a dilated and softened heart. The increased gravity of the case does not however depend upon the actual presence of the dropsical effusion, but because the existence of dropsy indicates that congestion of the systemic capillaries has reached its limits, or that visceral obstruction has attained an extreme degree. If we expect therefore to remove the dropsy, our efforts must first be directed to the removal of the congestion on which it depends, and to the relief of obstruction in the hepatic, and renal systems. Our efforts in those cases are however always crowned with success, the dropsical effusion removed, is certain sooner or later to return; still if the patient's constitution is not broken down, if he had not led an intemperate life, if his blood is not deteriorated, if he can command a regular exercise, and other accessories which his condition requires, the disease may be considerably postponed.

When the renal secretion can be increased, there is

\* Treat. on Disease of the Heart.

† Dublin Quart. Journal

better mode of diminishing or removing dropsical effusion than by the administration of diuretics. In general however the congested state of the kidneys prevents the elimination of watery fluid by them, and, "a serious error would be committed if we at once endeavoured to act upon them." "Their state of congestion cannot (Dr. Chevers observes) be relieved so long as the liver and heart are gorged with blood, and the skin remains inactive, or while the organs themselves actually suffer from the pressure of large ascitic effusion; under these circumstances all diuretics become local poisons." "The safest and most physiological course to consider to be, first to endeavour to restore and to excite the action of the skin." "A well-managed plan of active diaphoretic treatment can now scarcely be attended with danger—at the same time, mild expectorants and aperients may be employed, not with a less sanguine hope of present success. A strict limitation of ingesta will tend to relieve the heart, and to reduce the hepatic congestion, and then as the skin begins to act freely, and dyspnoea and palpitation becomes somewhat abated, a certain amount of relief to the kidneys may be anticipated, provided the congestion of these organs is not associated with advanced structural disease. This tendency to restoration of function may be aided by local abstraction of blood, the application of heat, counter-irritation, dry-cupping, &c."

In this stage of the disease, and when this combination of symptoms exists, we are accustomed to rely rather upon mercury carried to the extent of slightly effecting the gums, than upon diaphoretics. Indeed after local bleeding, there is no more effectual means, and sometimes it is the only one we possess, of relieving pulmonary and hepatic congestion, and of promoting the absorption of dropsical effusion. It has however several drawbacks, thus it is liable to run off by stool, and unnecessarily to weaken the patient; in other instances, and particularly if Bright's disease of the kidneys is present, it is liable to cause sudden, and profuse hæmaturia, instead of exerting any salutary effect upon the dropsical effusion; besides a mercurial course leaves after it more or less debility, and tends to induce a state of anæmia, the state of all these things which we would wish to prevent.

When *hypertrophy* predominates, and the general health is not much deteriorated, hydragogue cathartics are occasionally given

with considerable present relief. When *dilatation* much predominates, however, they are, as a general rule, inadmissible; they debilitate the patient without affording corresponding relief, and their employment is not without hazard; indeed, uncontrollable diarrhoea is occasionally the consequence of the imprudent use of medicines belonging to this class. As constipation is however often present, particularly when the liver and intestinal mucous membrane are both congested, an occasional dose of some of these medicines may be necessary; and, if the biliary secretion is scanty, a mercurial purgative is occasionally interposed with advantage.

The employment of the remedial measures which have been alluded to, is attended with much less satisfactory results in hospital, than in private practice. Indeed in the class of patients whom dropsy, depending upon this cause, sends into our hospitals, we can in general do little more than palliate the more urgent symptoms, sometimes not even so much. Insufficient nourishment, exposure to cold and hardship, not unfrequently combined with intemperance, have all had a share in undermining the patient's health; and the consequence is that dropsy supervenes earlier in such subjects, and when it does set in, not only are our means of relieving it more limited, but it is almost certain to recur sooner, owing to the continued operation of the same causes which originally produced it.

After a time, which varies in different subjects, and after perhaps the dropsy had been relieved and had recurred several times, a period invariably arrives (unless the disease and the patient's life had been cut short by some acute attack) when medicines cease to act, the dropsy increases despite the best directed efforts, and there is scarcely a more pitiable condition, or one more distressing to witness than this. Owing to the unwieldy state of the lower extremities the patient is almost helpless; incapable of lying down, he is obliged to pass the greater part of the night in a chair, or he is restless, moving from his bed to a chair, and from the chair back to bed. He obtains scarcely any sleep, and this only in the most constrained posture, and he looks forward with dread to the night; if he falls asleep he is quickly awakened by dyspnoea, or cough. The respiration is laboured, and accomplished with effort; the dyspnoea is constant, but recurs likewise in paroxysms of extreme

y, and is aggravated by the most trifling exertion. He is tormented from pain, sometimes very severe in the præcordial region, and has stitches in different parts of his chest. He is teased with cough, and sometimes there is considerable difficulty in expectoration.

The urinary secretion is scanty, and the free egress of urine is prevented perhaps by the œdematous state of the prepuce and scrotum. He is tormented with thirst, but afraid to drink, and his distress is often aggravated by flatulent distension of the stomach, and intestines.

In the latter length, the integuments of the lower extremities being stretched to the utmost, the cuticle cracks, or is elevated into blisters, which give way, and allow the dropsical fluid to escape; in other cases the surface becomes hot and painful, and a violent erythematous redness overspreads it, which ends in gangrene; the urine dribbling over the scrotum and upper part of the thighs, causes excoriation, and superficial sloughs of these parts, and the patient dies of exhaustion, or he goes off in one of the fits of convulsions, or he becomes comatose. Sometimes diarrhœa supervenes, which runs him down, at others he dies asphyxiated; occasionally he passes off quietly and rather suddenly, when apparently better than usual. The death is sometimes preceded by delirium, and often by unusual restlessness and irritability.

## CHAPTER XVIII.

**FATTY DISEASE OF THE HEART—FORMS OF—HYPERTROPHY OF THE CARDIAC FAT—FATTY DEGENERATION—SOFTENING OF THE HEART—CAUSES OF FATTY DISEASE—SYMPTOMS OF—PHYSICAL SIGNS—GENERAL SYMPTOMS—TREATMENT OF FATTY DISEASE.**

MORBID deposition of fat occurs under two very distinct forms in the heart; in one the normal amount of fat upon the heart is in excess, and this may not only coat the entire surface of the organ, but it may encroach upon its muscular tissue, and penetrate between the muscular fibres; in the other fatty matter is primarily deposited in the interior of the sheaths of the muscular fibres. In the first form, the muscular fibres are obscured, or atrophied by the encroachment of fat deposited outside them; in the second form, the muscular fibre itself undergoes fatty degeneration. In the one, the diseased condition is evident to the unassisted eye; in the other, the microscope is necessary to determine its presence. In the one, the morbid deposit commences at the pericardial surface of the heart; in the other it is usually first observed at the endocardial surface. The one condition under the name “adiposity of the heart,” has been long familiar to pathologists; our knowledge of the other is of much more recent date. To the former, Dr. R. Quain, in his memoir,\* which contains the best account of the disease we possess, suggests that the term *fatty growth on the heart* should be limited; to the latter, that of *fatty degeneration*. The term, *hypertrophy of the cardiac fat* being more expressive, will be used here for the first form.

Rokitansky makes three divisions of fatty disease of the heart; his two first forms are, however, merely different degrees of hypertrophy of the cardiac fat; but the terms he uses are calculated to lead to confusion. Thus, he applies the terms “actual fatty degeneration of the heart,” and “fatty metamorphosis” to fatty

\* Med. Chir. Trans. vol. xxxiii.

growth on the heart in its advanced stage; terms, which should be restricted to the cases in which the sheaths of the muscular fibres are the seat of the fatty deposit.

## HYPERTROPHY OF THE CARDIAC FAT.

The heart of the infant at birth is destitute of fat; in the adult subject, however, except in individuals dying of chronic disease attended with considerable emaciation, a certain amount of fat is usually found upon the surface of the organ, which increases as life advances, and, in corpulent persons is commonly present in considerable quantity.

This fat, which is identical with adipose matter in other situations, has its seat under the investing membrane, and in the first instance always occupies the grooves of the heart—as it increases, is observed in addition upon the thin edge of the *right* ventricle, upon its anterior surface, and apex, and upon the margin of the auricular appendages, particularly the left; it is less frequent on the surface of the left ventricle. When moderate in amount, it does not constitute a state of disease, and is not incompatible with the perfect performance of the heart's functions. When, however, it extends beyond these limits, when it encroaches upon the muscular tissue, penetrates between the fibres, and causes atrophy of the latter, it constitutes a serious form of disease. The muscular fibres are compressed and atrophied, they become paler than natural, and the tissue of the heart is lax and flabby. A section of the parietes of the affected chamber in extreme cases appears sometimes to consist of little else than fat; a thin lamina of muscular tissue next the endocardium only existing; or the entire thickness of the parietes of a portion, or of the entire of one of the chambers (particularly of the right ventricle) may be made up of adipose tissue, and the chamber may be opened without the division of almost any muscular fibres. In a few instances it has been observed to form little prominences in the interior of the ventricles, elevating the endocardium.

The adipose matter is contained in cells, analogous to fat cells in other situations; it has also the ordinary yellow colour of fat, but when the affection is advanced, and fat encroaches upon the muscular fibre, it is “less consistent, more oleaginous,” and has a

duller hue. It is met with more especially in individuals who have, in popular language “an unhealthy disposition to grow fat;” fat abounds in the anterior mediastinum, and upon the outside of the pericardial sac; the origin of the large vessels is surrounded with it, and the omentum and mesentery are usually loaded with it.

Hypertrophy of the cardiac fat may co-exist with fatty degeneration, or with other morbid conditions of the heart, or it may be independent of any. The muscular tissue of the heart may be atrophied in this condition, while the size of the entire organ is often increased; and although it is most commonly observed in corpulent persons, it occurs also and in a very marked degree in thin, or emaciated individuals. Out of 2161 autopsies recorded by Dr. Chambers\* in his *Decennium pathologicum*, excess of cardiac fat was met with forty-nine times; it was associated with chronic disease of the heart in thirty-two, in the seventeen remaining cases, no other disease of the organ existed. This excess of fat on the heart was associated with general corpulence in twenty subjects, and occurred twenty-nine times in persons not corpulent; but then, the whole number of corpulent persons examined was only 104, while the number of persons not corpulent examined, amounted to 2057.

The following table, given by Dr. Chambers, shows the morbid states of the heart which were associated with excess of its adipose tissue in the forty-nine cases :

State of the heart associated with excess of adipose tissue.	In corpulent persons.	In persons not corpulent	Total
Dilatation . . . . .	13	5	18
Hypertrophy . . . . .	...	6	6
Dilatation and hypertrophy . . . . .	2	3	5
Softening, or atrophy . . . . .	1	2	3
Healthy state . . . . .	4	13	17
Total . . . . .	20	29	49

FATTY DEGENERATION OF THE HEART.

Fatty degeneration of the heart was unknown as a distinct form of disease, or was described as a variety of softening, or was confounded with fatty growth on the heart until a comparatively recent period. We are indebted to Drs. Duncan and Cheyne,

\* Brit. and For. Med. Chir. Rev.



ums in these countries, and to Laennec, and Rokitansky  
ient, for the earliest notice of the subject; but, it is to  
Dr. Ormerod,† and Dr. Quain,‡ that we are princi-  
ed for our knowledge of its clinical history and pa-

generation is much more common in the ventricular  
icular portion of the heart, and in the left ventricle  
ht. It is usually present in different degrees in the  
and in its most intense form is commonly limited to a  
1 of the heart's tissue. It ordinarily commences in  
muscular fibre next the endocardium, and the fleshy  
the mitral valve are often implicated. It may also  
t the pericardial surface, and in extreme cases "it  
: entire thickness of the walls of the ventricle." It  
litary lesion, but in many instances it is associated  
eposit in other situations; as with atheroma in the  
h hypertrophy of the cardiac fat, with fatty degene-  
cornea, or with fatty disease of the liver, or kidneys.  
rist with any morbid condition of the heart; as with  
f endo or pericarditis, with dilatation, or hypertrophy  
ricles; or, with disease of other organs, as chronic  
nd emphysema of the lungs, phthisis, cancer, Bright's  
neurism.

, in 143 hearts from subjects dying of various diseases,  
or less fatty degeneration in 100; a fatty state of the  
sociated with sixty-eight of the latter. The other  
which it was most frequently combined were

				Cases.
culosis ...	...	...	...	21
ed heart	...	...	...	31
ed kidneys	...	...	...	38
ed liver	...	...	..	3
onia ...	...	...	...	8
utis ...	...	...	...	7
and spinal cord	...	...	...	7
with a fibrinous diathesis	...	...	...	13
ions or accidents followed by bronchitis, or				
alirium tremens ...	...	...	...	15

generation of the heart is not only a very formidable

fed. Gaz. Aug. 1847.      ‡ Med. Chir. Trans. vol. xxxiii. 1850.  
. 1849.

lesion *per se*, but it constitutes a very serious complication if the patient becomes the subject of another disease, particularly pneumonia, fever, acute bronchitis, cholera, erysipelas, or delirium tremens; or if an accident or other cause renders a capital operation necessary. Indeed, it is not improbable that in some of the cases in which typhus fever proved unexpectedly fatal, the death was owing rather to the patient's heart having previously been the seat of fatty degeneration, than to the occurrence of softening as the result of the fever.

Fatty degeneration of the heart is always accompanied by alterations in the colour, and microscopic appearances of the tissue of the part, generally by alterations in its consistence, and sometimes also by alterations in the size of the organ.

*The Colour* of the heart varies in different cases, and in different parts of the same heart, according to the stage, and extent of the disease. As a general rule, the degenerated muscular tissue is paler, and has a duller hue than natural; often with a buff, yellow shade, similar to that of faded or withered leaves, to which Laennec compared it; or more that of a fatty, or boiled liver. In other cases, the tissue has a dirty brown or leaden hue, resembling wet brown paper; and, occasionally it presents, when cut or torn, a granular aspect, like the lung in an early stage of granular hepatization, to which the term "granular degeneration" has been applied by Mr. Paget.

The alteration in colour is not uniform throughout; "it is generally (Dr. Quain observes) in spots, or patches; and though the whole heart may be pale, the spots being more so, give the tissue, when seen beneath the endocardium, a mottled or spotted appearance." "The spots are of infinite variety of size and form, such as lines, crescents, circles, &c. As the disease advances, the spots run together, and give portions of the heart a uniform buff coloured appearance. This appearance may be confined to a portion of the heart, the rest presenting the ordinary healthy characters."

These alterations of colour can be recognised by the unaided eye, but it requires the microscope to determine the changes which have taken place in the degenerated muscular fibre.

*Microscopic appearances.*—When a portion of the degenerated tissue is submitted to a high power of microscope, the transverse

striae of the muscular fibrils are found to be indistinct, or absent; and their sheaths are seen to contain very minute oil globules, which may be few and scattered, or so crowded as to fill the sarcolemma, according to the stage the disease has reached. The oil globules at first have the appearance of minute black dots, "which partially obscure the proper substance of the fibre. As these dots grow larger so their centres grow bright, and they manifest themselves as particles of oil, which still increasing both in size and number almost completely fill the sarcolemma."

These oil globules are always much smaller than those in fatty growth on the heart, their size "rarely exceeds that of the blood-corpuscles; while that of the black dots may be in the first instance not more than one tenth of the measurement." "They resemble (Dr. Quain observes) the oil globules of milk;" and "that they are composed of fat is demonstrated by their highly refractive properties, and by the action of ether upon them when they have escaped from the sarcolemma."

When the tissue of the heart has undergone this change, the fibres as a necessary result become friable, and break down readily; "the proper muscular tissue at the part (as Mr. Paget remarks) is gone, and in its place we have tubes full of fatty matter, therefore powerless, and wholly incapable of exercising the proper function of the muscular fibre."

*The consistence* of the heart when the seat of fatty degeneration is, as a general rule, altered; it becomes soft, flabby, or friable, according to circumstances. In the slighter forms, however, and when much healthy tissue is mixed up with the degenerated fibres, the heart may be in no appreciable degree softer than natural; on the other hand, it may be flabby, and flaccid, without much fatty disease being present. "This flaccid state appears (Dr. Ogle observes) to be rather dependent on imbibation, and a fluid state of the blood, as in fever, puerperal peritonitis, pyæmia, &c." When fatty degeneration is advanced, the tissue of the heart, in addition to being softened, becomes friable, and tears upon a slight force; and in extreme cases, the muscular substance breaks down under the pressure of the fingers, like wet brown paper; the scalpel at the same time is greased in cutting it, and blotting paper pressed upon it gets a greasy stain.

A heart thus affected undergoes decomposition more quickly

than a healthy heart; and, there is sometimes a rapid evolution of gas, which distends the superficial veins soon after death, a point first noticed by Professor Smith. This very advanced condition of the disease is seldom general, as usually met with, it engages but a limited portion of the heart's tissue.

*Alterations of size.*—A heart, the seat of fatty degeneration, may be enlarged, or it may be smaller than natural, or it may preserve its normal dimensions—it is most frequently enlarged; thus in eighty-three cases tabulated by Dr. Quain, the size of the organ was increased in forty-three, and diminished in only four. This increase in size is due to *dilatation* of the ventricles, which may be associated with either hypertrophy, or attenuation, or with fatty growth on the heart. According to Rokitsansky, fatty degeneration occurs more especially in hypertrophied and dilated hearts. He regards it as “a consecutive disease of hypertrophy of the heart; developed in consequence of the state of paralysis, or inertness of certain portions of the muscular substance, induced by the disproportion between the mass of the tissues and the powers of innervation.” It appears to me that dilatation is rather a consecutive result of softening; the softened walls of the ventricle yielding to the distension from within, and being incapable of returning to their normal state owing to the degeneration of the muscular fibre, distension passes into dilatation, which becomes permanent.

The condition of the muscular fibres of the heart in fatty degeneration has been shown by Dr. Quain to be analogous to the peculiar chemical change that takes place in dead animal tissues, when exposed to moisture and excluded from the air, and known under the name of *adipocire*. The change has been regarded by some as identical with that which occurs in voluntary muscles when long unused, as in old unreduced dislocations, ankylosis of joints, &c.; but the two conditions are not analogous, “the tendency of voluntary muscles (as Dr. Quain remarks) is to degenerate into a fibrous tissue mixed with fat, rather than into granular fatty matter.”

#### SOFTENING OF THE HEART.

We have seen that fatty degeneration is accompanied by softening, and friability of the tissue of the heart; and it constitutes the most frequent cause of softening. Writers generally, however, have treated of softening as if it were a distinct form of disease;

and have made divisions, and sub-divisions according to the different ideas they entertained respecting its nature. Most of these varieties would more properly come under the head of fatty disease; in which category may probably be included "the whitish and yellowish softening" of Laennec and Hope; the "cachectic softening" of Pigeaux, the "senile softening" of Bland, and some of the varieties made by Andral. There are, however, a certain number of cases, in which softening of the heart is independent of the conversion of its tissue into fat; among which may be included, the softening that occasionally occurs in typhus fever, in bad forms of scurvy, in chlorotic anæmia, purulent infection and puerperal peritonitis.

The softening of the heart which sometimes occurs in the latter states, equally as that which attends upon scurvy and chlorotic anæmia, being but a part of the constitutional disease, it is not necessary to delay upon them here. The variety which is occasionally observed in typhus fever demands, however, some notice. This form of softening was first mentioned by Laennec, it has been particularly described by Louis;\* and its clinical history has been investigated with much care by Dr. Stokes.†

According to Louis, softening of the heart is more common when the fever proves fatal early; in seven instances noticed by him (where the softening was extreme), death took place before the fourteenth day of the fever. The softening was very rarely limited to the left side of the heart, it was generally universal, though sometimes more marked in the left ventricle. The heart was flaccid, in many "it retained, like a wet cloth, any shape in which it was placed;" it was also friable, "easily torn, and very easily penetrated by the finger." The size of the heart was not increased, it appeared smaller in two of his cases; and, in nearly all, the walls of the ventricles, particularly of the left, were attenuated.

The appearances of the heart in this morbid state, according to Dr. Stokes,‡ are as follows:

"The heart is little, if at all, altered in volume. It is generally of a livid hue, but this it may have in common with other internal organs, as is often seen in fever. It feels extremely soft

\* Recherches sur la Gastro-enterite.    ‡ On Diseases of the Heart and Aorta

† Dub. Jour. of Med. vol. xvii.

especially in its left portions, and the left ventricle frequently pit on pressure. Nothing remarkable is to be found as to the pericardium or endocardium, and the valves are unaffected. The principal change is found in the muscular structure, which is often infiltrated with an adhesive, as it were, gummy secretion. The left ventricle exhibits a singular appearance, for the traces of muscular fibre are lost, and the external layer, to the depth of the eighth of an inch, converted into a homogeneous structure, in which no fibre can be found. The colour of this altered portion is generally dark, and it resembles the cortical structure of the kidney. In some cases this change occurs in patches, varying in depth, and from a quarter to three quarters of an inch in breadth. The change may affect the septum cordis and, but to a much less degree, the right ventricle. The internal net work of fleshy bundles appears less engaged, and though these may be pale, their firmness seems but little altered. The right ventricle is almost always more firm and hard than the left, which may be so softened as to break down under a slight pressure."

"In most of the cases in which signs of softening of the heart were observed, the patients exhibited a dark-coloured and abundant petechial eruption; the mouth was covered with sordes; the body exhaled the peculiar foetor of typhus, while extreme prostration and stupor were generally present. In a large proportion of cases the secondary bronchial disease existed to a great degree, coming on in a singularly latent manner at about the fifth day of the affection, and although without much cough, or prominent dyspnoea, often advancing so as to endanger life. The gastro-enteric symptoms were generally less developed, though not unfrequently present. In many instances the disease occurred in young and robust men, and the bad symptoms were developed at an unusually early period; the petechial eruption and the prostration appeared soon, and crisis was rare, yet the convalescence was generally satisfactory, and the restoration to health complete. The cases were, in other words, well-marked examples of the typhus of this country, and in a large number the disease could be traced to contagion. The health of these patients, previous to the attack, was in nearly every instance, good. There was no appearance of the scorbutic condition, and in the fatal cases it was almost always found that the voluntary muscles were red, showing the cadaveric rigidity well-marked, and in no respect diseased."

the anatomical and vital phenomena of this affection . Stokes observes,) to the opinion that it is an example of special secondary lesions of typhus, like the infiltration of the lymphatic glands of the intestine ; and capable of retrocession and consequent disorganization."

## CAUSES OF FATTY DISEASE.

hypertrophy of the cardiac fat, and in fatty degeneration of the heart we have examples of a constitutional tendency to abnormal deposition, or abnormal deposition of fat ; and the term *fatty* might with as much propriety be applied to such constitutions, as the terms *scrofulous* or *gouty* are, where the diseases are marked. The fatty diathesis, then, may be, 1. a constitutional tendency to excessive secretion, or deposition of fat, either in *abnormal quantity*, or in *abnormal position* ; in the former it is deposited in abnormal amount in places where it is *normally* present ; in the latter it is deposited in places where it is *not normally* present. Of the former, we have examples in excessive corpulence, in excessive deposit of fat on the heart, in the omentum, mesentery or other parts. Of the latter, we have examples in fatty degeneration of the heart, in fatty deposit (atheroma), in the large arteries, in fatty disease of the eye, in the arcus senilis of the cornea, &c.

In the heart we have, as has been said, examples of each. The mere presence of fat in excess does not constitute an actual state of disease, but the presence of the latter even in moderate amount is a serious condition. When excessive, it may occasion atrophy of the muscle in the vicinity of the part in which it is deposited ; and in some cases is an actual conversion of the muscular fibre into fatty tissue. In the former there is simply hypertrophy of the cardiac muscle, and in the latter there is a conversion of albuminous into oily matter. In other words, of a highly organized material into one of a lower scale of organization, and it constitutes therefore a true disease. The former is met with in youth, in the adult, and in old age ; the latter is rather a disease of the decline of life, and is more frequent in old age.

The fatty degeneration of the heart, by modern authorities, is often regarded as a form of *atrophy*. Undoubtedly atrophy of the muscle is an effect of excessive fatty deposit on the heart ; and,

without any necessary alteration of its tissue. Indeed *atrophy* is generally understood as the very opposite *trophy*; and if applied to an hypertrophied heart (affected with fatty degeneration not unfrequently is), it is calculated to lead to confusion.

#### CAUSES OF HYPERTROPHY OF THE CARDIAC FAT.

Hypertrophy of the cardiac fat is frequently associated with general corpulence, though it is met with likewise in individuals who not only are not encumbered with superfluous fat, but are absolutely lean. Its original source is of course the food. The blood is known to contain a certain amount of fat (about two parts in a thousand); it does not, however, exist in a free state in this fluid, and cannot be detected by the microscope. In certain diseased states, however, it has been found in a free state; thus the opaque or milky appearance of the serum of the blood, in certain cases has been shown by Dr. Traill to be due to the presence of oil globules; and Professor Smith† has described two cases of fatty degeneration of the heart, in which he found oil in the blood.

Hypertrophy of the cardiac fat is met with in corpulent individuals who have passed the prime of life, and who lead indolent

\* "The blood, in a state of health, contains three distinct kinds of fat:

"1. Saponifiable fats composed of the spermaceti-like substance called stearin, which is also found in the blood.



individuals generally who live well, and take little exercise. The fatty matter of the aliment being rapidly assimilated, is deposited on the heart equally as in other situations.

It is occasionally also met with in individuals who are temperate in their habits, neither eating or drinking to excess, and who are lean rather than corpulent. It will generally however be found that such persons seldom take exercise sufficient to promote perspiration; they limit themselves either to passive exercise, which as is little better than passive; and the fatty constituents of food which find their way into the blood, not being consumed in the lungs, or not passing off with the perspiration, are retained, and deposited as cardiac fat.

Latham thinks, it is not in individuals who have been long thin, but that this morbid deposition of fat is to be apprehended, when a man has been always thin, and in the decline of life suddenly becomes fat."

Hypertrophy of the cardiac fat is met with also in individuals who make an habitual free use of malt liquors, or who have been addicted to ardent spirits in excess, and such persons may be well nourished in other respects, or absolutely lean. Here it is the excess of hydro-carbon in the drink, the blood contains an excess of fat, which is deposited upon the heart in inconceivable amount; or, owing to disease of the liver induced by the same habits, a cachectic state is brought about, and "fatty degeneration is formed at the expense of albumen and fibrin, by a process of emulsification." Indeed M. Huss\* has shown, that, the blood of individuals in the constant habit of using spirituous liquors to excess

"contains a much greater abundance of oily or fatty matter than in the blood of abstemious persons; in fact, the blood of drunkards is saturated with carbon, and at the same time the fibrin and globules diminish."

#### CAUSES OF FATTY DEGENERATION.

Fatty degeneration of the heart may almost always be traced to imperfect, or deficient nutrition, or to disordered or depraved habits; which itself may be the result of *constitutional* or of *acquired* causes. It is important to bear this distinction in mind, as it has considerable influence upon the complications of the disease. For instance, when the cause is constitutional, fatty

\* Brit. and For. Med. Chir. Rev. April, 1852.

degeneration of other tissues is frequently associated with it; of which *atheroma* in the large arteries, and the *arcus senilis* are common examples; while a considerable portion of the muscular tissue of the heart is usually engaged. Whereas, when the cause is local (as obstruction or obliteration of a coronary artery), fatty degeneration of other organs, is not so likely to be associated with it, and the disease is more limited; while its progress is generally more insidious, and it is more liable to terminate in rupture of the parietes.

Fatty degeneration of the heart depending upon constitutional causes, may not unfrequently be traced to a morbid state of the blood, which is either impoverished, or vitiated; and this may be the result of previous disease, acute, or chronic, particularly the latter, if of long standing and attended with exhausting discharges; or when the individual has been bed-ridden for many months, or crippled by rheumatic arthritis, or other causes. It is sometimes the ultimate result of deficiency of food, unhealthy habitation, prolonged mental toil, or anxiety, intemperance, &c., in fact anything which interferes with nutrition, or impairs the general health, may prove a cause.

Derangement and impairment of nervous influence, by interfering with nutrition, favour the occurrence of fatty degeneration. It is well known that paralyzed muscles waste, and eventually undergo alteration; and Rokitansky attributes much influence to this as a cause of fatty degeneration of the heart.

Mental anxiety by interfering with nutrition indirectly proves a cause of fatty degeneration "by withdrawing the body from those exercises which constitute main sources of its vigour; by depressing the heart's action so that the blood is circulated with insufficient frequency, and force; by modifying the respiration so that the oxygenizing of the blood is less perfect than it should be; by interfering with the digestive function; and, oftentimes by banishing or breaking sleep."\* Dr. Quain met with it several times in individuals who had suffered much mental distress; in three such cases, the degeneration led to rupture of the heart.

Advanced age appears to be a predisposing cause, partly owing, as Mr. Barlow observes, "to the impaired condition of the blood, partly to the withdrawal of nervous influence, and partly to

\* On Fatty Degeneration, by the late W. F. Barlow.

the enfeeblement of the vital functions, digestion, respiration, and circulation." A gouty tendency may often also, as Dr. Ormerod remarks, be traced to the subjects of it. Fatty degeneration of the heart, although most frequent in the decline of life, is by no means limited to that period; it has been observed under puberty and at every period afterwards.

In determining the influence of age in its production, we should be led to form an erroneous estimate, if we limited ourselves to the cases, in which death was directly the result of the fatty degeneration of the heart; the majority of such subjects having been advanced in life; whereas, when death was the result of other diseases, and only perhaps indirectly hastened by the state of the heart, the subjects were generally much younger. Thus in one hundred hearts, examined by Dr. Ogle,\* from patients dying of various diseases, which all presented more or less of fatty degeneration, he found that in twenty-three, marked as only very slightly fatty, the average age was forty years; while in nine, marked as decidedly fatty, the average age was thirty-six. The highest age attained in the latter class was sixty, and one was only ten years of age.

When fatty degeneration depends upon a *local cause*, it is generally such as interferes with the coronary circulation, and prevents a portion of the heart's tissue from receiving the supply of arterial blood necessary for its nutrition. This may be the result of endocarditis, ending in adhesion of a semilunar valve to the inner surface of the aorta, or of adventitious deposit around the root of the aorta, obstructing or occluding the orifice of one of the coronary arteries; or of the same deposits within the tube of a coronary artery, diminishing its calibre, and impeding the passage of the blood through it. In the two latter instances the fatty degeneration of the muscular fibre is often a *secondary* effect of fatty, or other adventitious deposit in the vessels supplying the heart. The frequency of these as causes of fatty degeneration is shown by Dr. Quain's tables;† from which it appears that out of eighty-three cases, the coronary arteries were ossified or obstructed in twenty-five; and valvular disease, chronic endo, or pericarditis existed in seventeen.

When the circulation in one coronary artery is obstructed, the

\* Trans. Path. Soc. vol. iv.

† Med. Chir. Trans. vol. xxxiii.

effects upon the heart are more prejudicial than would be the case in a similar obstruction of an artery in other parts of the body, owing to the nature of the anastomoses of the coronary arteries; a point to which attention was first called by Mr. Swan.\* He has shown that “although the coronary arteries communicate at the base and apex of the heart, the communication is not very free, and each can do very little more than supply its respective regions.” Hence if one coronary artery is partially or completely obstructed, the part of the heart, to which its branches are distributed cannot receive an adequate supply of blood by the anastomosing branches, its tissue consequently will be insufficiently nourished, and degeneration may ensue.

Atheromatous, calcareous, or other deposit in the coronary arteries, when in an extreme degree, by interfering with the nutrition of the heart, may, instead of giving rise to fatty degeneration, bring about *atrophy* of the entire organ. Tiedemann† has recorded a remarkable case of this kind, where the heart weighed only two ounces three drachms, and measured but two inches seven lines in its long diameter. The patient was a female, seventy years of age, both coronary arteries were ossified, and the orifice of the right so much contracted as hardly to admit a probe. Dr. Ogle‡ mentions another, but in his case the heart weighed seven ounces.

In several of the cases, in which ossification of the coronary arteries was present, symptoms of angina pectoris were observed; indeed this condition of the arteries was supposed by Jenner to be the cause of angina, and Parry, Black, and others have recorded examples of it. It is, however, only indirectly a cause, inasmuch as it usually leads to degeneration of the heart's tissue.

#### SYMPTOMS OF FATTY DISEASE.

The effect of hypertrophy of the cardiac fat when in excess, or of fatty degeneration, being to diminish the contractile force of the ventricles, and to impair their power of propelling the blood, the impulse becomes more feeble and indistinct, or is absent; the pulse becomes weak, intermittent, irregular or slow; the sounds of the heart diminish in intensity, or one, rarely both may be inaudible.

\* Lond. Med. Gaz. Nov. 1848.

† Trans. Path. Soc. vol. iv

‡ On Arctation and Closure of Arteries.

As the enfeebled ventricle is incapable of fully expelling its contents, distension of its chamber is liable to ensue, followed sooner or later by permanent dilatation. The brain, under those circumstances, not receiving its normal supply of blood, or the circulation in this organ being otherwise disturbed, syncopal attacks ensue in some subjects, and semi-apoplectic seizures in others, one or other of which the patient may die; or upon some slight muscular exertion, the parietes of the weakened ventricle may yield, and give way—the blood escapes into the pericardial sac, and the patient dies.

In considering the symptoms of fatty disease it should be borne in mind that they must vary according to a variety of circumstances, as the extent of the disease, and its complications; but, particularly its seat, whether on the left or the right side of the heart, and its age, whether early or advanced. Thus, if the right ventricle alone is engaged, it can exercise no direct influence upon the radial pulse; whereas when the left ventricle is its seat, the pulse presents certain well-marked characters. Again, when the right ventricle alone is its seat, those syncopal attacks observed in certain cases are less likely to occur, but by the tendency of this state of the ventricle to impede the return of the venous blood from the brain, it may assist in inducing the semi-apoplectic seizures which are occasionally observed in this diseased state.

#### PHYSICAL SIGNS OF FATTY DISEASE.

*Impulse.*—In fatty disease of the heart, when at all advanced, the impulse, as a general rule, becomes weaker than natural, and eventually imperceptible both to the eye and hand in its normal position. A patient under my care had himself noticed this disappearance of the heart's pulsation, and remarked on it as extraordinary. If the right ventricle does not participate in the disease, the impulse will be perceptible to the right side and about the sternal cartilage, or sometimes below it. When the ventricles are hypertrophied, the impulse may not be diminished, or although ordinarily weak, it may become temporarily strong under excitement. In the advanced stage of fatty degeneration, or when it is complicated with dilatation and attenuation, the impulse besides being very feeble, becomes more or less irregular.

In *softening of the heart* in typhus fever, “the loss of impulse

find it by examining when the patient is turned on his back, or by pressing with the fingers in the intercostal spaces at the end of expiration, when we discover it like a feeble vibration. In most cases, the diminution of impulse is attended by a corresponding loss of sound; but it must not be forgotten that impulse and sound are not always proportionate or connected in the invasion, or retrocession of the disease."

*Sounds of the Heart.*—The sounds of the heart are diminished in intensity, particularly the first sound, which is almost extinct; in a few instances, the second sound has been very feeble, while the first was well-marked. If the disease is limited to the left ventricle, the sounds will be louder on the right than the left side of the præcordial region. The pulse, and the action of the heart are slow, both sounds are perfectly distinct, slow, and measured, with a long interval equalling the period occupied by the double sound. In a case under my care, where, in addition to slow pulse, jugular pulsation was present in both the internal and external jugular vessels appeared to be double that of ordinary systole, as if a more feeble ventricular systole intervened between ordinary systole. Fatty disease of the heart, *per se*, does not give rise to a murmur; and, if valvular disease is associated with it, the physical signs of the latter will be less marked than when muscular tissue is sound.

The examination of the heart in tubercular fever is the same

at both sides of the heart, is indicative of an extreme weakening."

#### GENERAL SYMPTOMS OF FATTY DISEASE.

*Pulse.*—Three distinct characters of pulse are met with in disease of the heart.

small, feeble, and compressible pulse, which may be or intermittent.

very small, intermittent, irregular, and unequal pulse.

permanently slow pulse.

The effect of fatty disease being to diminish the propulsive force of the left ventricle; we should expect to find the pulse small, feeble, smaller, and more compressible than natural in such cases, and this is exactly what does generally occur; a strong irregular pulse being incompatible with a softened left ventricle. It occasionally happens, however, that the radial artery at the wrist, feels hard and firmer than natural, owing to ossific deposit in the vessel, when this condition of the artery is met with and other signs of fatty disease are marked, ossific deposit in one or both arteries is probable. As the morbid condition advances, the pulse, in addition to being small, becomes intermittent, and very irregular, and in extreme cases, it is so small, irregular, and unequal as to be with difficulty counted, when it resembles the pulse of extreme contraction of the mitral orifice. This condition of pulse may be the result either of fatty disease *per se*, or of a combination of dilatation, or attenuation of the left ventricle in fatty disease.

In softening of the heart from other causes, the pulse presents the foregoing characters, being either small and feeble, or intermittent and irregular. Indeed, an intermittent or irregular pulse was long upon as a very unfavourable sign in fever, before it was known to be connected with softening of the heart. Louis was the first to notice the latter; he states, that, in almost all the cases in which this character of pulse was present, and the patient died, the structure of the heart was found to be softened.

*Pulse.*—In certain cases of fatty heart, the pulse, instead of presenting the foregoing characters, is permanently slower than natural, being sometimes only from twenty to forty in a minute,

or less. The connection between slow pulse, and fatty heart, was first noticed by Dr. Macbride,\* of Dublin. "The accumulation of fat about the basis of the heart, and the large vessels connected therewith, incumber it (he says), so that neither the systolic nor subsultory motions can be performed with sufficient freedom whence the weakness and slowness of the pulse."

In some cases of this disease the pulse presents this feature to a very remarkable degree; thus, in one, quoted in Mr. Richardson's valuable memoir† upon "Slow Pulse," it was for a time so low, nine in the minute; and in another, also quoted by him, it was for a time only eight in a minute. Mr. Richardson has also made the remark, that a sudden rise in the frequency of the pulse sometimes occurs in these cases shortly before death. According to Dr. Todd,‡ the pulse, in feebleness of the heart, may be more frequent in the recumbent than the erect posture, contrary to what holds good in health: this, however, is by no means a general rule. In a case under my care, where the pulse in the recumbent posture was thirty-six, it was not increased in frequency on making the patient sit up; on another occasion, when the pulse of the same patient was thirty-eight in the recumbent posture, it rose to forty-four in the sitting posture. It may also happen in cases of this kind; that while the action of the heart is slow, the pulse itself is so very feeble as to be imperceptible or nearly so at either wrist; this, however is rare.

When a permanently slow pulse is met with in one class of cases, and a very small, irregular, and unequal pulse in another; it is scarcely necessary to say, there must be some cause for this remarkable difference. Dr. Stokes attributes the slow pulse to an "equally weakened state of both ventricles;" but the opposite form of pulse is observed here, as often as a slow pulse. Dr. Ormerod thinks, that a slow pulse is characteristic of his second form of fatty degeneration; but, it is met with in both forms, as when both are combined.

It may happen, that the slowness of the pulse is only apparent and depends upon every second systole of the left ventricle being too feeble to communicate an impulse to the blood in the radial artery. When, however, there are no reasons to suppose this

\* Practice of Physic, 1772, p. 217.

† Lond. Med. Gaz., vol. xiv.

‡ Dub. Quarterly Journal, vol. xiv.



be the case, we must look for another explanation. If the cases of fatty disease are analysed, in which a slow pulse was a prominent symptom, they will generally be found to have been attended by symptoms of disturbance of the cerebral circulation, indicated by fits of weakness, or faintness, by syncopal attacks, or by semi-apoplectic seizures; and it is to the deranged or disturbed state of the circulation in the brain that the slowness of pulse is due, not to differences in the seat or nature of the fatty disease. Indeed, I have observed the speech to be slow, and the patient generally lethargic, when the pulse was permanently slow. Mr. Richardson\* has also made a somewhat similar remark. "With regard to the slow pulse sometimes met with in these cases, I think (he says) we must look to the nervous system and not to the heart for its explanation, for in different persons, with this same disease of the organ, and in the same stage of the disease, the pulse varies from extreme rapidity to remarkable slowness."

*Dyspnœa* may be absent altogether; or it may be experienced only on ascending stairs, or in making some muscular exertion, or it may be the symptom especially complained of by the patient. In some instances it is constant, in others it occurs in paroxysms, particularly at night, often coming on during sleep and without apparent cause. The latter varieties depend often upon the association of chronic bronchial, or pulmonary affections with the fatty disease. In uncomplicated cases the sensation is rather one of faintness, or giddiness, experienced in close or crowded rooms, or brought on by slight mental excitement; or, it is more a feeling of breathlessness, which causes the patient to pant or to sigh frequently. Sometimes it is accompanied by a choking sensation, or by a sense of constriction or tightness across the chest, with occasionally a feeling of sinking. The respirations may, or may not be more frequent than natural; in a case under my care, where the pulse was only thirty-three in the minute, the number of the respirations was precisely the same.

Dr. Stokes has described a form of "respiratory distress," which he considers to be peculiar to this disease, "consisting of a period of apparently perfect apnœa, succeeded by feeble and short respirations, which gradually increase in strength and depth, until the respiratory act is carried to the highest pitch of which it seems

\* Dub. Hosp. Gaz., May 1855.

of; it is sometimes, however, a prominent and distressing  
it is usually referred to the præcordial region, or to the  
to a point behind the sternum, or to the epigastrium, from  
it radiates to the axilla, left scapula, and arm.

The sensation experienced at first, is a dull uneasy feeling  
amounting to pain, which is either constant or intermittent  
over a space which can be covered by the palm of the hand  
referred to the region of the left nipple, or to the left of  
As the disease advances, the uneasy sensation passes into pain  
increases in severity at times, and is not affected by pressure  
generally by a full inspiration. In other instances the pain  
the first is paroxysmal, the patient being free from it in  
vals, the paroxysms are sometimes of extreme severity  
the characters of *angina*; the pain is referred to the præcordial  
præcordial regions, from which it shoots to the left scapula  
extends down the left arm, or sometimes down both arms  
paroxysms at first are few and far between, ultimately  
become frequent, or may even occur daily. The first attack  
often follows some unusual exertion, or some mental distress  
In several instances where pain was not experienced until  
short time before death, it was connected with rupture of the  
fibres of the heart; and then was sometimes referred to  
the epigastric region. From an abstract of eighty-three cases  
by Dr. Quain, it appears that long-continued pain was ex-

the fit, as occurs not unfrequently after ordinary syncope ; nor has he the sensation of noise, or buzzing in the ears, which is usually experienced in passing into or out of a fainting fit. One patient who was not insensible in the fit, described the sensation on coming out of it as being agreeable rather than otherwise ; another who was insensible in the fit, thought on recovering from it that he had been asleep and dreaming. Some, however, have described the sensation as a very unpleasant one, and while it lasted they have been impressed with a feeling of impending death.

These attacks are usually of short duration, occur at irregular, sometimes distant intervals, at others in quick succession ; they are most common at night and generally supervene without any warning, though in some instances they were preceded by vomiting, giddiness, faintness, or oppression of breathing. They are often brought on by something which taxes too much the powers of the weakened heart, whether mental or otherwise ; or, by the use of food which the stomach is unable to digest, either from its bulk, its kind, or its imperfect mastication.

In the cases in which these syncopal attacks were marked, the left ventricle was almost always the seat of the fatty degeneration. Their cause appears to lie in the blood being propelled to the brain with insufficient force, or in insufficient amount to maintain the *vascular pressure* essential to the due performance of its functions. Dr. Burrows\* has shown that the suspension of the functions of the brain which occurs in ordinary syncope, is the result of "insufficient vascular pressure on its substance, rather than of an inadequate supply of blood to the organ." When the left ventricle is softened, both these may come into operation, and it is easy to understand that the cerebral circulation may be readily deranged under these circumstances from very trifling causes. Dr. Stokes mentions a case where "on the occurrence of the premonitory symptoms, the patient by hanging his head so that it rested on the floor" was able to ward off an attack. These attacks are more liable to occur when fatty degeneration supervenes upon hypertrophy of the left ventricle ; because here the vessels of the brain, which previously had been constantly fully distended, are now no longer so, and the "vascular pressure" is diminished in proportion.

Syncopal attacks are always unfavourable symptoms, the disease

\* On Disorders of the Cerebral Circulation.

is necessarily somewhat advanced when they first set in; the patient ultimately may die in one, or they may be succeeded by semi-apoplectic seizures, or alternate with the latter. In the cases tabulated by Dr. Quain, death by syncope is stated to have occurred in thirteen out of thirty-three in one series, and in eight out of thirty-five in a second. In the thirteen cases in the first series, both sides of the heart were affected in five cases, the left side in seven, the right in one. "This mode of death is in many cases (Dr. Quain observes) instantaneous; in other cases, death, though sudden, is not so rapid in its occurrence, the fatal faintness is progressive, and death may not occur for several minutes after its onset."

*Semi-apoplectic seizures.*—When fatty disease is advanced, it is sometimes attended by transient fits of coma, which come on without any warning, or are preceded by giddiness, or a feeling of faintness, are rarely attended by stertor, by convulsive movements of the limbs, or true convulsions, and are not followed by paralysis. The duration of the fits may be only a few minutes, or longer; they occur at irregular intervals, sometimes during sleep, and they set in at a more advanced period of the disease than the syncopal attacks, which may or may not precede them. During the fit, the patient is quite unconscious, the face is pale, the pupils are unaltered, and the surface is cold. The return of consciousness is sometimes marked by slight convulsive movements, rarely by frothing at the mouth; the patient for a short time after the fit is sometimes lethargic, and his memory appears impaired.

That coma, whenever it occurs, is the result of pressure on the brain, admits, I believe, of no doubt; and that the cause of the coma here must be transitory, is proved by the shortness of the fits, and the perfect recovery from them within a short period. In the case before us, we have a feeble left ventricle, feebly propelling the blood to the brain, and incapable therefore of causing overdistension of its vessels. We may, however, have combined with it a softened state of the right ventricle which would tend to induce venous congestion of the brain. The latter is sufficiently common in the advanced stage of obstructive valvular disease, but the symptoms which accompany it have little resemblance to those we are considering. We must therefore look to some other explanation for the comatose symptoms; the cause of which probably

lies in the varying amount of the cerebro-spinal fluid, which, as Dr. Burrows has shown, is "supplemental to the other contents of the cranium, at one time giving place to an increased quantity of blood in the cranium, at another making up for a deficiency of blood in the vessels in the head."

When the blood has been for some time transmitted to the brain in diminished amount, or with diminished force, we would expect the cerebro-spinal fluid to be increased in quantity to compensate for the deficiency of blood in the cerebral vessels. Now if, from any cause, this fluid cannot readily change its position upon any temporary obstruction to the return of the venous blood from the brain, the cerebral substance will be exposed to increased pressure, sufficient to cause temporary coma. After a short time this ceases spontaneously, or, under the influence of stimulants, temporary strength is given to the heart, and the attack ceases. Death by coma is not very common; in the eighty-three cases tabulated by Dr. Quain, seven only died by coma.

*Arcus senilis*.—Mr. Canton was the first to demonstrate\* that the arcus senilis is a fatty degeneration of the cornea, and to call attention to its frequent association with fatty disease of the heart. Those who have seen this state of the cornea frequently associated with fatty heart, have set it down as a symptom of the latter; while others, to whom it has not so often presented itself, deny that it is to be regarded as a diagnostic sign. Dr. Williams† says he has "found it present in about nine-tenths of the cases in which he had reason to infer the existence of fatty degeneration of the heart," and he has given a table‡ of twenty-five cases in which the arcus senilis, in a more or less marked degree, was present, and the prominent symptoms were such as are usually present in fatty disease. Another table of twelve cases has been given by Dr. Hoskins,§ in which the arcus senilis was present, without there being any reason to suspect fatty degeneration of the heart—"only two of these patients laboured under symptoms referable to the heart, and in only one could any organic alteration of the organ be suspected. Six, or one-half, were healthy individuals, and one was a young man under thirty, of remarkable health and

\* The Lancet, vol. i. 1850.

† The Lancet, 1850.

‡ Principles of Medicine, 1856.

§ Amer. Jour. of Med. Sci., Jan. 1853.

vigour." Mr. Barlow\* mentions an instance where the arcus senilis, "which had been both well and frequently observed, disappeared."

We have already said that the arcus senilis, and fatty disease of the heart are each signs of the *fatty diathesis*, the local deposit having its seat in the heart in one, and in the cornea in the other. The arcus senilis, consequently, is no more a symptom of fatty heart, than fatty heart is a symptom of arcus senilis; the two deposits are distinct and independent of each other; and may, or may not be associated in the same subject. When fatty degeneration of the heart is the result of constitutional causes, the arcus senilis is more likely to be present than when it is the result of a local cause, such as obstruction, or obliteration of a coronary artery. Its presence, therefore, is not an evidence that the individual labours under fatty heart; nor is its absence by any means an indication that he is not the subject of fatty disease of the organ.

*The Countenance* in fatty degeneration of the heart is usually pale or sallow, the pallor in the advanced stage being occasionally quite remarkable, and the same bloodless condition of the rest of the surface is observed. The hair is usually gray, and in the advanced stage is, not unfrequently, perfectly white. The individual is more frequently lean than fat, or he is "fatty rather than what is usually understood by fat." The extremities, owing to the feeble circulation are usually cold, and the individual is generally very sensitive to cold; and before the disease has made much progress, he remarks himself that he is easily fatigued, or that he perspires upon slight exertion.

#### TREATMENT OF FATTY DISEASE.

In considering the treatment of fatty disease, a distinction must be drawn between the cases in which we have to deal simply with hypertrophy of the cardiac fat, and those in which fatty degeneration exists. Excess of cardiac fat may be absorbed, and unless it has penetrated between the muscular fibres, and caused ulterior changes in them, the affection may be regarded as remediable; but when the muscular fibre itself has undergone degeneration, and these fibres are filled with oil globules in place of their normal

contents, no medicine, or plan of treatment known, can restore them to their original state of integrity.

#### TREATMENT OF HYPERTROPHY OF THE CARDIAC FAT.

The treatment of hypertrophy of the cardiac fat, supposing it to be accurately diagnosed, would be pretty nearly the same as for general corpulence, with which it may, or may not be associated. If we can succeed in causing absorption of the subcutaneous fat, we may hope to bring about absorption of some of that which encroaches upon the tissue of the heart.

To carry out this object, diet and regimen are more to be depended upon than medicines; though there are several of the latter which enjoy reputation in this respect. As far as diet is concerned, fat, butter, oil, milk, saccharine and farinaceous substances, malt and spirituous liquors should be avoided or sparingly used. Dr. Begbie\* in his notice of the case of the late Dr. Thalmers (whose death was the result of fatty disease of the heart), mentions that "the continued use, for a few weeks together, of a mixture of spirits and water with sugar, was invariably attended in him with a manifest increase of subcutaneous fat, which subsided on the disuse of the stimulating drink."

In the kind, and amount of exercise, we must be guided by the stage the disease has reached, and by the patient's ability to undergo it; when it is deemed advisable it should be carried out regularly and systematically, but cautiously; and whether active or passive, it should be short of inducing dyspnœa. Dr. H. Kennedy,† who is an advocate for exercise, observes, "in the treatment of such cases, and with the hope of curing them, bodily exercise, suited to each individual case, and carried out with a specific end in view, ought to take a prominent place." Among medicines, both alkalies and acids have had advocates; to produce such effect, however, they require to be persevered in for a long time; but they have the disadvantage of being liable to derange the digestive organs when administered in this way. In carrying out the treatment "we must not forget," Dr. Chambers observes, "that, fat on the heart, whether it be the last to come, or not, will rarely be the last to go; and we must carry on the plans which we have found beneficial beyond the diminishing of the abdomen

\* Ed. Month. Jour. vol. xii. 1851.

† Dub. Med. Press, vol. xxii. 1849.



and limbs, if we would relieve the over-burdened centre of circulation."

Hypertrophy of the cardiac fat does not often, however, give rise to symptoms calculated to attract attention until the tissue of the heart has been encroached upon by the fat, and its muscular fibre has undergone attenuation or atrophy. The treatment then merges into that of fatty degeneration, with which it is sometimes associated.

#### TREATMENT OF FATTY DEGENERATION.

Fatty degeneration of the heart must be regarded as an incurable affection; we cannot restore muscular fibres which have been spoiled, or replace them by healthy tissue, and all that we can hope to accomplish may be summed up in a few words, viz.:

1. To palliate or remove pain, dyspnoea, and other distressing symptoms.

2. To endeavour to prevent the disease from increasing or extending.

3. To endeavour to prolong life, and to render it more endurable.

If there is one morbid condition more than another, in which dietetic and hygienic rules require strict attention, it is that we are considering. The food should be nourishing, easily digested, and not of a kind to occasion flatulence; watery fluids should be sparingly used, but a certain amount of stimulants, either wine or brandy is indispensable; the food should be properly masticated, and its bulk should never be such as to overload the stomach. From inattention to these rules, trifling as they may appear, syncope or apoplectic seizures and death have ensued. Constipation should be strictly guarded against; in several instances, rupture of the weakened ventricle has occurred in straining during the act of defecation. Strong mental emotion of every kind is to be avoided; many examples are on record where a state of excitement was the immediate cause of death. The amount of exercise whether passive or otherwise, should be strictly limited to the capabilities of the patient; if the disease is advanced, he cannot be too guarded in this respect, and should confine himself entirely to passive exercise. The heart, though damaged, may be equal to performing its functions as long as the circulation is tranquil; but



the additional strain put on it by a sudden effort, or muscular exertion may prove too much for it, and has often eventuated in rupture. Hence the patient should not be exposed to the necessity of ascending flights of stairs, but should have his bed-room and sitting-room upon the same floor. The body likewise should be warmly clothed, and the cutaneous circulation promoted by friction, &c.

The more purely medical part of the treatment consists in the administration of medicines calculated to relieve pain, or dyspnoea; to shorten the fits of syncope or coma, or to remove their effects; to improve the quality of the blood, and to invigorate the general health.

When *pain* is severe, occurs in paroxysms, and presents the characters of angina, we must have recourse to opium, which here is advantageously combined with diffusible stimulants, and antispasmodics. The liquor opii sedativus is the most generally useful form; the solution of muriate of morphia comes, perhaps, next. The opium may be combined with chloric or sulphuric ether, or with the aromatic spirit of ammonia, and given in camphor mixture. I have used chloroform internally, but the patients always experienced more relief from opium. At the same time counter-irritation may be employed, or liniments containing ludanum, aconite, or chloroform, may be applied to the painful part. Sometimes two or three leeches to the præcordial region, by assisting in relieving distension of the ventricle, give temporary ease; they cannot however be repeated often, patients labouring under this form of disease bear loss of blood badly. When any doubt exists as to the propriety of applying leeches, dry cupping may be substituted for it.

In the syncopal attack, the patient should be placed in the recumbent posture, the dress loosened, air freely admitted, the face sprinkled with cold water, smelling salts applied to the nostrils, and some stimulant given (if the patient can swallow), either brandy, or ether, with aromatic spirit of ammonia. Heat should be applied to the præcordial region, and if the fit continues, turpentine, or a mustard cataplasm should be applied to the same part. In a case quoted in Mr. Richardson's memoir, the treatment ultimately found to answer best in the fit was "simply to fan the face, apply eau de Cologne to the nostrils, forehead, and

temples, and, if there was a disposition to a continuance, to give tea or coffee."

In the intervals between the anginal, or syncopal attacks, or in their absence, tonics in combination with one another, or with medicines belonging to other classes are generally of service; and among these, the preparations of iron and quinine are the most efficacious. At the same time, derangements of the digestive organs are to be attended to, constipation is to be prevented, and the biliary and cutaneous secretions are to be maintained in as healthy a state as possible.

With the view of strengthening the weak heart, Dr. Law has recommended strychnine in minute doses; I have employed the extract of *nux vomica*, with this object, in the dose of a third of a grain twice a day, and certainly with some advantage. The nitric, or nitro-muriatic acids in some instances have been found by Dr. Williams\* advantageous; in others ammonia; and "one is tempted (he says) to attach some meaning to the peculiar composition of these agents, as being of an opposite nature to fat, the acids affording abundance of oxygen, which may remove a part of the superfluous fat, and both these and ammonia supplying azote, which may contribute to the formation of a more highly animalized plasma."

The treatment of softening of the heart must vary with the cause which has given rise to it. Thus, in fever, when the pulse and cardiac phenomena indicate weakness of the heart, the period for the exhibition of wine (as Dr. Stokes has shown) has arrived. When softening supervenes upon scurvy, anæmia, &c., the treatment must be directed to the primary disease; when it occurs in convalescence from acute affections, it is to be treated as a disease of debility. In other cases, the treatment must have reference rather to the complications.

*Inhalation of Chloroform.*—The state of fatty degeneration of the heart constitutes a strong objection to the employment of chloroform in surgical operations, death having ensued in several instances where it was used even in moderate amount. In some of the cases, the chloroform, no doubt, was exhibited in the sitting posture. When anæsthesia supervenes in this position, and the contractile power of the heart is diminished, owing to the degener-

\* Principles of Medicine, 1856.

on of its tissue; the weakened ventricle may be incapable of propelling the blood to the brain, and death by syncope may ensue. When, however, the inhalation was used in the recumbent posture, the immediate cause of death was probably failure of the coronary circulation, owing either to impediment to the return of the blood to the coronary veins, or to impure blood being conveyed to the tissue of the heart by the coronary arteries, or to both combined.

For instance, in this state of the heart, the weakened left ventricle, on anæsthesia supervening, is liable to become over-distended, when the coronary equally as the general circulation will be impeded, and the blood accumulating at the right side of the heart, the coronary veins will be unable to empty themselves. The irritability of the weakened heart being, at the same time, still further impaired by imperfectly oxygenized blood being conveyed to its tissue, we cannot be surprised if its movements sometimes suddenly cease under these circumstances.

That inhalation of chloroform is liable to interfere with the arterialization of the blood, is evidenced by the fact, that, in operations performed under its influence, this fluid as it escapes from the divided arteries, has sometimes all the sensible qualities of venous blood. Hence great caution is necessary in employing it in this condition of the heart; "an agency powerless over a healthy and vigorous organ, is quite sufficient (as Dr. Quain observes) to overwhelm the feeble and tottering action of a heart that is diseased."

## CHAPTER XIX.

CARDIAC ANEURISM—FORMS OF—ANEURISM OF THE LEFT VENTRICLE—ANEURISM OF THE LEFT AURICLE—ANEURISM OF THE CORONARY ARTERIES—CAUSES OF CARDIAC ANEURISM—COMPLICATIONS OF—SYMPTOMS, PROGRESS, AND TERMINATIONS OF.

By the term cardiac aneurism, or aneurism of the heart, we understand a partial dilatation of the parietes of one of the chambers of the heart, limited in a great measure to the left ventricle; which resembles, in some respects, aneurism of the large arteries, and like it may end in rupture; but in most instances terminates fatally without bursting. This diseased state attracted little attention until the appearance of Breschet's\* memoir upon the subject, notwithstanding that several well-marked examples of it had been placed on record by Morgagni, Walter, Baillie, Corvisart, Berard, and Mr. Adams of this city. Breschet's memoir was followed in 1829 by one by Reynaud;† but Mr. Thurnam's‡ paper, which appeared in 1838, contains the fullest account of the disease we possess. His was followed in 1843 by a very excellent memoir on the same subject by Dr. Craigie;§ and in 1846, by one by Dr. Peacock.|| Cruveilhier, Johnson, Elliotson, Bouillaud, Rostan, Hope, Rokitansky, Hasse, and others have also contributed to our knowledge of its pathology.

Cardiac aneurism occurs either with, or without distinct external tumor, the latter being the most common. When distinct tumor is present, this may consist of a simple bulging of a portion of the parietes, or the latter may form a sac communicating with the chamber by a narrower orifice. The tumor may be no larger than an almond, or it may equal the heart itself in size; the latter however is rare. It is usually solitary, but several have been met with in the same heart.

\* Report Gen. d'Anat. tom. ii.

† Journal Hebdomad, Feb. 1829.

‡ Med. Chir. Trans. vol. xxi.

§ Ed. Med. and Surg. Jour. vol. lix.

|| Ibid, vol. lxvi.

The site of cardiac aneurism is almost always the left ventricle, but a few cases have been recorded where the left auricle was its seat. Every part of the left ventricle is not equally liable to be the seat of aneurism, it is most common at the apex, next at the base, and then between the apex and base; it has rarely been observed in the septum; out of fifty-eight cases noted by Mr. Thurnam, it occurred only three times in this situation; since then, Drs. Craigie and Pereira have each recorded a case. The walls of the aneurism vary in thickness at different parts, when the sac is large they are usually thinner than under opposite circumstances. Membranous plates are occasionally developed in the walls of the sac. The interior may contain stratified layers of fibrin, or they may be absent; when the aneurism is sacculated, and of long standing, fibrin is more commonly found in it, than when the aneurism is saccular and not sacculated.

#### FORMS OF CARDIAC ANEURISM.

According to Mr. Thurnam, the several forms of cardiac aneurism have their counterpart in aneurism of the large arteries. The following are the sub-divisions made by him:

1. When the parietes of the aneurism are formed by the pericardium alone, or with some of the outer layers of muscular fibres, after rupture or ulceration of the endocardium, and inner layers of muscular fibres; analagous to *false aneurism* of the arteries.
2. When the parietes of the aneurism are formed by all the coats which enter into the parietes of the ventricle; analagous to *true aneurism* of the arteries; which is the most frequent.
3. When the entire circumference of a portion of the ventricle is engaged in the dilatation, Mr. Thurnam terms it *diffused true aneurism* of the heart, from its analogy to cases where the entire circumference of an artery is dilated.
4. When the aneurism forms a canal under the lining membrane of the ventricle, which opens at some other point, he terms it *dissecting aneurism* of the heart.
5. When the lining membrane protrudes through the ruptured muscular tissue of the ventricle, and forms the aneurismal sac, it may be said to be analogous to the *aneurisma herniosum* of authors.
6. Finally, "in the case of an aneurism in the septum ventriculorum becoming ruptured so as to form a communication with a

portion of the venous system—the right ventricle—we have (he says) a lesion, altogether analogous to that which results from the wound of an artery, and its accompanying vein, to which the name *spontaneous varicose aneurism of the heart* is perfectly applicable.”

*Aneurism of the Left Auricle* was first described by Dr. Elliotson,\* who has given a drawing of the morbid appearances. Aneurism in this situation is usually nothing more than dilatation either of the sinus, or appendix of the auricle, generally the latter; it is almost always associated with contraction of the mitral orifice, and the dilatation is more diffused than in aneurism of the ventricle, usually involving the entire sinus, or appendix. “The dilated walls of the cavity are often (Mr. Thurnam observes) thickened, and the seat of fibro-cellular degeneration. The lining membrane is opaque, rough, and otherwise diseased, and in some cases, even ossified, and is lined with fibrinous layers, very similar to those met with in arterial aneurism. In all the cases, the lining membrane appeared to be continued into the interior of the dilated part, which consequently merits the name of true aneurism.” When the dilatation engages the auricular appendage, its cavity is sometimes nearly obliterated by fibrinous deposit.

Sacculated aneurism of the left auricle is extremely rare, but three examples of it being on record, one by M. Chassaignac,† another mentioned by Rokitsansky, which was an example of the acute form, and was seated in the auricular septum. The third has been recorded by Mr. Fenwick,‡ the aneurism in this case sprung from the body of the auricle, was of the size of a small orange, and the case is remarkable for the absence of disease of the mitral valve.

*Aneurism of the Coronary Arteries* constitutes another but a very rare form of cardiac aneurism, five examples only being known. The earliest on record was given by Hedlund, and is referred to by Otto,§ it occurred in a man of forty years of age, and proved fatal by rupture into the pericardium. Within a few years, two have been recorded in the Transactions of the Pathological Society of London, one by Dr. Peacock,|| the other by Dr. Bristowe.¶

\* Lumleyan Lectures, Fol. 1830.

† Revue Medicale, 1839.

‡ The Lancet, Jan. 1846.

§ Path. Anat. trans. by South.

|| Trans. Path. Soc. vol. i.

¶ Ibid, vol. vii.

The aneurism may be seated in either the trunk or branches of the coronary arteries; it is usually solitary, but in Dr. Bristowe's case numerous aneurismal dilatations existed in both the branches and trunks of these vessels. It varies in size from that of a pea to a pigeon's egg; it may or may not contain laminated fibrin; and may terminate by rupture into the pericardial sac, or the patient may die without the occurrence of rupture. It may be associated with atheromatous or calcereous deposit in other parts of the coronary arteries, or on the valves of the heart; and in the few examples on record, no symptom, during life, led to a suspicion of the existence of the disease. In Dr. Peacock's case, a peculiar pinging sound, distinct from the cardiac sounds, loud along the sternum and towards the left side, was heard the day before the patient's death, and he complained of pain in the præcordial region.

In this case, however, traces of recent pericarditis were found, attributed probably by the irritation of the growth of the tumor. Dr. Peacock's and Dr. Bristowe's patients were both males, the former, whose age was fifty-one, had been in the habit of lifting heavy weights, and was very intemperate; the latter was a sailor, aged twenty-two, and there was no cause to which the disease could be referred.

*Aneurism of the Valves* has been already considered under the head of valvular disease.

Rokitansky\* is of opinion that every variety of cardiac aneurism may be included under one of two heads, viz., *acute* and *chronic*; the former corresponding to the *false* aneurism of Mr. Burnham, the latter to the *true*.

The *acute form*, according to him, is the result of acute inflammation of the endocardium and innermost layers of muscular fibres, inflammatory softening ends in laceration or rupture of the endocardium; the blood entering the fissure distends the pericardium and adjacent muscular fibres, and a cavity is formed, "which is surrounded at its mouth by a torn and fringed margin of endocardium." This form of aneurism is much more rare than the other, is developed rapidly, and is accompanied by signs of recent endocarditis; it is met with in the young and old, and never attains as large a size as the chronic form. In all the cases seen by Rokitansky "the aneurismal formation was of recent origin, and

\* Path. Anat., trans. by Day, vol. iv.

wall of the heart throughout the whole of its thickness, as by endocarditis and pericarditis." "The inflammatory cellular substance, by its tendency to induration, promotes the formation of a white fibroid tissue, which takes the place of the cellular fibre, and coalesces internally with the thickened myocardium, and externally with cellular or fibroid formations, the products of endocarditis and pericarditis." This tissue, being capable of resisting the pressure of the blood, yields at the point of rupture, and thus gives rise to a circumscribed dilatation at the point of rupture.

The comparative frequency of these two forms of aneurism is very different, the chronic or true aneurism being much more common. Thus out of twenty-eight cases referred to by Thurnam, in which this point is noticed, the dilatation in two was formed by all the structures entering into the wall of the ventricle; and in six only was there a solution of continuity in the lining membrane, and internal layers of muscle, either as a consequence of ulceration or, what is more common, rupture.

#### CAUSES OF CARDIAC ANEURISM.

According to some authorities cardiac aneurism is the result of inflammation; others, with more reason, will admit the influence of inflammation in its occasional production, but consider that it is more often the result of non-inflammatory causes.



arterial system, that it is scarcely ever due to a solution of continuity in the lining membrane of the heart. Undoubtedly, when it attains a large size and assumes a sacculated form, the endocardium and muscular fibres may be partially absorbed and disappear, and it may assume the appearance of false aneurism; but it is also certain, that cardiac aneurism scarcely ever occurs in a heart, the parietes of which are otherwise perfectly sound, the muscular tissue at the part is flaccid, softened, or degenerated; or, the endocardium and the endocardium have undergone changes the result of antecedent inflammation. Atheromatous deposit is occasionally seen in the interior of the ventricles, but cardiac aneurism is seldom or never the result of this deposit and of its subsequent softening.

What Rokitansky terms the *acute form* of cardiac aneurism might, I think, to be included under the head of *ruptures of the heart*; it is in fact a partial or incomplete rupture of the parietes of the ventricle, the endocardium and innermost layers of muscular fibre being softened as the result of acute inflammation, or other cause, give way; the pericardium and outer layers of muscular fibres next yield to the distending force of the blood, and a partial dilatation ensues; the latter in their turn are ruptured and death ensues within a comparatively short period.

The cause of aneurism of the left auricle, appears in general to be in the impediment to the passage of the blood from the auricle into the ventricle, owing to narrowing of the orifice of communication between the two chambers. As contraction of the mitral orifice is a very common lesion, Mr. Thurnam is of opinion "that many of the cases which have been reported as exhibiting simple dilatation of the left auricle in connexion with it, were really cases of aneurismal dilatation."

The immediate exciting causes, according to Mr. Thurnam, were, in one, an injury of the chest, in another, a violent fit of anger, in one (a private soldier), retention of the breath during military flogging, and in another, mental anxiety. In Mr. Fenwick's case of aneurism of the auricle, lifting a heavy sack of salt upon the shoulders appeared to be the exciting cause; the patient at the moment having experienced a sudden pain in the chest, accompanied by faintness, and sickness.

## COMPLICATIONS OF CARDIAC ANEURISM.

Cardiac aneurism is scarcely ever a solitary lesion, other parts of the heart being almost invariably likewise diseased, and it may be associated with almost every morbid condition of the organ. Thus it is sometimes accompanied by the results of inflammation in the endocardium, valves, or pericardium; at others by dilatation with or without hypertrophy of the ventricles, or by some alteration or degeneration of the muscular tissue of the walls of the organ. The endocardium may be thickened, or opaque; and ossific deposit is common upon the aortic and mitral valves. The parietes of the left ventricle are sometimes partially converted into "a yellowish white nearly homogeneous mass, resembling boiled white of egg;" at others the tissue has a darker hue than natural; the substance of the walls of the left ventricle, in a few cases was indurated, at others softened. Dilatation of the left ventricle, or of the other chambers of the heart, usually with hypertrophy, was present in most of the cases on record.

Cardiac aneurism may be associated with old general adhesion of the pericardium, or with partial adhesion at the site of the tumor, or with recent lymph upon the pericardial surfaces. The latter is not met with unless the aneurism forms a distinct tumor upon the surface of the heart; nature appears to endeavour to strengthen its parietes by setting up pericarditis at the part, which ends in adhesion between the opposed layers of the membrane. In a case which was under my care, where the aneurism was seated at the apex of the ventricle, the pericardial surface of the heart was coated with recent lymph at the period of the patient's death (which arose from other causes), pericarditis having apparently been called into action by the enlargement of the aneurismal tumor. In a case recorded by Mr. Adams, the pericardium was not only adherent, but the sac had, in addition, contracted adhesions with the central tendon of the diaphragm below, and anteriorly with the pleura, the cartilages of several of the ribs, and the intervening muscles.

## SYMPTOMS OF CARDIAC ANEURISM.

In none of the cases of cardiac aneurism which have as yet been recorded, was a diagnosis made during life; the disease was discovered only upon post-mortem examination. In some of these

cases, no symptom referable to the heart was present during life, and when well-marked cardiac symptoms existed, other parts of the organ were generally so much diseased as to account for the symptoms present.

When cardiac aneurism is, as it were, embedded in the substance of the ventricular walls and does not form a prominence upon the pericardial surface, it can scarcely give rise to any physical or other sign, which would lead to a suspicion of its existence. When it projects beyond the surface, forming a moderate sized tumor, it may, as Breschet suggests, cause a dull sound to be yielded by the parietes of the thorax towards the left side inferiorly; and may also modify the impulse and sounds of the heart.

When the sac attains a considerable size, projects much beyond the surface of the heart, and has a narrow neck, it may not only interfere with the movements of the organ, but a bruit of some kind would probably be audible with the first, or second sounds of the heart, or with both. In addition, if situated at the apex of the left ventricle, it might form a pulsating tumor externally; there is, however, no recorded case where this occurred; but, in a very remarkable one given by Mr. Adams,\* where the aneurismal sac nearly equalled the heart itself in size, it approached the surface in the fifth and sixth intercostal spaces, and the tumor here was covered only by the integuments and intercostal muscles, which latter had become pale and thin. If the sac was situated near the base of the ventricle, and was of such a size as to compress the parts in its vicinity, it might obviously simulate aneurism of the ascending portion of the arch of the aorta.

We are indebted to Mr. Thurnam for the best account of the symptoms which may lead to a suspicion of the existence of cardiac aneurism.

*Physical signs.*—In three of the cases collected by him, the impulse of the left ventricle was increased; in one, the action of the heart generally was forcible and tumultuous; and in two it was feeble and obscure. In four cases, including two of the above, a murrells or rasping sound was heard with the ventricular systole, and in another, a similar sound was audible to the left of the sternum. Dr. Peacock says,† that in four cases, M. Gendrin

\* Dub. Hosp. Reports, vol iv.

† Ed. Med. and Surg. Jour., vol lxvi.

observed a murmur with the impulse of the heart, ceasing abruptly with the systole, and followed by an interval of silence, after which a short diastolic murmur succeeded. In Mr. Fenwick's case, the first sound of the heart was accompanied by a bruit de soufflet near the apex, which was audible only over a space the size of a five shilling piece. In Dr. Craigie's\* case, in which an aneurism the size of a walnut was seated in the septum of the ventricles, the first sound of the heart was prolonged to double its usual duration, and was accompanied by a rough murmur, partaking of the characters of rasping and blowing. "While the cardiac pulsations were strong, forcible, and about from forty-four to forty-six in the minute, the arterial pulsations were disproportionately small and feeble; and occasionally one or two beats were omitted, or not transmitted to the artery, so that the arterial pulse was seldom above forty." In one of Mr. Thurnam's cases, where the action of the heart was feeble and obscure, the character of the first sound was short like that of the second. Dr. Hope has recorded a case where "steatomatous degeneration had caused the formation of a canal from the aorta underneath one of the sigmoid valves, and the internal membrane of the left ventricle, leading to an aneurism as large as a nut, in the substance of the auriculo-ventricular septum;" in which the second sound of the heart was accompanied by a murmur.

*Local and General Symptoms.*—In many of the recorded cases *pain* was complained of, but its character and seat differed; occasionally it was very slight, merely a sense of uneasiness; at other times it was severe, resembling that of angina, or aneurism of the aorta; while in some it was only complained of on pressure. In the majority, the pain was referred to the præcordial region, and was increased by anything which hurried the heart's action; occasionally, the chief pain was referred to the epigastric region. Mr. Thurnam thinks that an oppressive sense of heaviness or weight in the præcordial region, will be found to be one of the more constant of the symptoms which belong to aneurism of the heart, in its advanced stage.

*Palpitation* was a prominent symptom in nine out of twenty-three cases noted by Mr. Thurnam: in Mr. Adams' case, the action of the heart was so violent as to be perceptible through the

\* Ed. Med. and Surg. Jour., vol. 59.

patient's clothes. *Dyspnœa*, "in several instances amounting to the severest form, or orthopnœa, was present in fifteen of these cases." The *pulse* was "feeble, sometimes in an extreme degree, and seven;" in the majority it was regular. "Anxiety, dread of death or restlessness were noted in eight cases; and syncope, or a disposition to it, in three." In addition to these symptoms, cough, throbbing of the carotid arteries, jugular pulsation, occasional redness or suffusion of the face, inability to lie in any position but on the back, epistaxis, hæmoptysis, and sudden starting from sleep were noticed in others. In Cruveilhier's case, in addition to severe asthmatic paroxysms with constriction about the heart, the patient complained of a peculiar sensation in the axillæ. In the two cases given by Dr. Latham, the attack which proved fatal was ushered in by vomiting and diarrhœa. Lastly, "dropsy more or less extensive supervened, in ten cases."

#### PROGRESS—DURATION AND TERMINATION.

In the great majority of cases, cardiac aneurism arises gradually and imperceptibly. When the symptoms set in suddenly, they are analogous to those of partial rupture of the ventricle, where the accident did not prove immediately fatal. Its duration is very variable, the acute form is occasionally very rapid, one case upon record having terminated fatally in ten days. In thirteen instances, which this point is noted by Mr. Thurnam, the period over which the disease extended varied from three or four months to sixteen years.

Cardiac aneurism is more common in the male than the female; of forty-seven cases referred to by Hasse, thirty-five were males and twelve females. It is more frequent in the adult than in early life; out of forty-two cases, thirty-two were over thirty years of age, and ten under thirty. The youngest patient in Mr. Thurnam's list was eighteen years of age, the oldest past eighty; Dr. R. Quain\* has since recorded a case where the patient was fourteen years old, and Rokitansky says he has observed the same form in childhood.

The termination of cardiac aneurism may be sudden or otherwise; it may be the result of rupture, or it may be due to the applications, or the patient may die of some other disease. Out

\* Trans. Path. Soc. vol. iii. 1850.

asphyxia, and in the remainder, probably from

When rupture occurs the blood generally pericardial sac, rarely into the pleura, and the pericardial and pleuritic adhesions existed. If seat of the aneurism, it may open into the right sionally the fatal event has been immediately rupture of one of the coronary veins involved example of which was communicated to the S Ireland, by Mr. Tufnell.\*

The death is not always sudden even when interval of several hours may elapse, if the aperture blood escapes is minute. Dr. Peacock† has received five days probably intervened between the pericardium and the death of the patient. In this case, adposed layers of the pericardium had existed, and blood went on but slowly, as the pericardial broken up.

With respect to treatment little need be added means of diagnosing the very early stage of the its advanced stage, supposing that a diagnosis was treatment would not differ from that of aneurism of the advanced stage of valvular or other organic heart.

\* Dub. Med. Press, May, 1850.

† Ed. Med. and

## CHAPTER XX.

RUPTURE OF THE HEART.—FORMS OF.—TRAUMATIC RUPTURE.—IDIOPATHIC OR SPONTANEOUS RUPTURE.—CAUSES OF.—SYMPTOMS AND DIAGNOSIS OF.

SOME of the morbid conditions which have been described, terminate occasionally in rupture of the heart; this accident may also be the result of external violence. The former come under the head of idiopathic, or spontaneous ruptures; the latter of traumatic; and these two forms differ not only in the cause which has given rise to the accident, but generally also in the part of the heart which is the usual seat of the rupture.

Rupture of the heart may be either *complete*, extending through all the tissues forming the parietes of the chamber; or *incomplete*, or partial, extending through a portion only of its parietes; or implicating simply a fleshy column, a tendinous cord, or the curtain of a valve. The latter have been already alluded to in the chapter upon valvular disease. The rent may be so small as to be with difficulty discovered after death; and its site is sometimes indicated simply by a little ecchymosed spot upon the surface of the heart. On the other hand, cases are recorded, where the ulceration reached nearly from the base to the apex of a ventricle; never however, in the idiopathic form at least, extends through the orifices, the tendinous structures being sufficiently resistant here, to prevent it. The rupture is generally solitary, but several are sometimes met with in the same heart: in eight cases of this kind referred to by Ollivier, there were two rents in some; others, as many as five; and rupture has occurred in both ventricles simultaneously. Sometimes the rent internally is single, while externally there is more than one; or, it may be very small internally and large externally. When the ventricles are the seat of the rupture, the external orifice is generally larger than the internal; though this is sometimes reversed.

## TRAUMATIC RUPTURE.

Traumatic rupture may be the result of *direct* violence, as a heavy weight falling upon the chest, the wheel of a loaded vehicle passing over it, a violent blow upon this part, as the kick of a horse, or a non-penetrating gun-shot injury. Or it may be the result of *indirect* violence, as a fall from a considerable height, the roof of a house, a scaffolding, &c. In either case, there may be no ecchymosis or other evidence of contusion externally; in the former, the sternum and ribs are not unfrequently fractured in addition; in the latter, some other internal organ, as the spleen, liver, or diaphragm, are occasionally also ruptured.

In traumatic rupture, the part of the heart which has the thinnest walls is usually the seat of the laceration, hence it is most frequent in the auricles and right ventricle. In eight cases given by Dezeimeris,\* the rupture was three times in the right auricle, twice in the left auricle, twice in the right ventricle, and once in the left ventricle. In five original cases recorded by Mr. Prescott Hewett,† the seat of the rupture was the left auricle in two, the septum in two, and the right ventricle in one. In traumatic rupture from direct violence, the laceration may be in any direction, and the auricles or right ventricle are its usual seat: when the injury is caused by indirect violence, as a fall from a height, the auricles, according to M. Pigeaux,‡ most frequently suffer, and they may be ruptured either at the base, or near the septum.

The subject of traumatic rupture has been recently investigated with much care by Mr. J. S. Gamgee,§ who has tabulated twenty-eight cases of this accident. From his table it appears that the cause of rupture was in ten, the passage of a wheel over the chest, or other forcible compression of the part; in nine, falls from a height; in four, the kick of a horse, and in four, bullets fired against the chest from a gun or pistol. The duration of life after the accident in twenty of these cases, was as follows: death was instantaneous in eleven, "one got up, ran a few steps, and fell dead, one lived half an hour, two lived two hours, one three hours, two four hours, and one (an infant) lived fourteen hours,"

\* Arch. Gen. de Med. tome v.

‡ Mal. du Cœur, tome i.

† Trans. Path. Soc. Lond.

§ Researches on Path. Anat. and Clin. Surg.



the rupture in the latter case being in the right auricle, and nine lines in length.

In twenty-one cases, in which the co-existing lesions are stated, the liver in three, and the spleen in three, were the seat of laceration. In six there was no bruise of the chest-wall; in three the bruise was slight; in eleven cases there were fractures of the ribs or sternum, or of both; in six the pericardium was likewise torn. The seat of the rupture in twenty-two of these cases was the right ventricle in eight, the left in three; the right auricle in four, and the left in seven. The accident is much more common in the male than the female; twenty out of twenty-five of the patients were males. The ages varied considerably, two were infants, and three seventy years of age.

#### IDIOPATHIC OR SPONTANEOUS RUPTURE.

The earliest case of spontaneous rupture on record, is one given by Harvey; Morand and Haller were the next to notice it; Morgagni, when he wrote, was able to collect eight cases, and Portal subsequently recorded three. It is remarkable that Baillie, in his long experience, met with only a single example, and Corvisart tells us that he never met with one, though he has recorded some of rupture of the fleshy columns, or tendinous cords of the valves.

Since then, the subject has been pretty fully investigated, and we are indebted to Rostan,\* Blaud,† Rochoux,‡ Townsend,§ Dezeimeris,|| Ollivier,\*\* and Pigeaux,†† for much of the information we possess respecting it.

Spontaneous rupture is most frequent in the left ventricle, next in the right ventricle, and is least frequent in the auricles. In forty-nine cases collected by Ollivier, the left ventricle was its seat in thirty-four, the right in eight, the right auricle in three, and the left in two. In fifty-four cases collected by Pigeaux,‡‡ the left ventricle was its seat in forty-four, the right in eight, the right auricle in one, and the left in one also.

The rent may be about the base of the left ventricle, close to the apex, or near the septum. In the above forty-nine cases, it

\* *Nouv. Jour. de Med.* t. vii.

† *Bibliothèque Med.*

‡ *Theses de Paris*, No. 215.

§ *Cyclop. of Med.* vol. iv.

|| *Arch. Gen.* t. v.

\*\* *Dict de Med*, t. viii.

†† *Path. du Syst. Circ.* t. i.

‡‡ *Jour. Hebdomadaire*, tome viii.

occurred nine times at the apex. It is more frequent upon the anterior than the posterior wall of this ventricle, and its site is often along the septum. The direction of the laceration is generally parallel to the muscular fibres, and in the long axis of the heart, more rarely transversely; in the auricles it may be either longitudinal or transverse. In forty-nine cases, in which this point is noted by Pigeaux, the long diameter of the rent was parallel to the fibres of the ventricle in thirty-six, and transversely in thirteen. The edges of the rupture may be smooth, or jagged, and irregular: the rent may be a simple fissure, or it may have more or less of a circular shape; and the walls of the ventricle have occasionally an ecchymosed appearance in the vicinity of the rupture. If the rupture has been gradual, and from within outwards, the blood may insinuate itself between the fibres of the ventricle, and give rise to the appearance which has been termed *apoplexy of the heart*.

Idiopathic rupture is more common in the male than the female, and much more so in advanced life than at any other period. In the majority of the cases on record, the age was between sixty and eighty, it is rare under fifty; indeed, M. Bland terms the accident *déchirement sénile du cœur*, from this circumstance.

Spontaneous rupture never, I believe, occurs in a heart the tissue of which is perfectly sound; and the morbid conditions of the organ upon which it most frequently supervenes, are:

1. Fatty degeneration of the heart—and softening of the organ from other causes.
2. Dilatation with or without attenuation of the ventricles—or congenital thinness or weakness of the parietes.
3. Obstructive valvular disease, when associated with either of the preceding morbid conditions.
4. Abscess, or ulceration in the parietes of the heart.
5. Aneurism of the ventricles.
6. A varicose, or other diseased state of the coats of the coronary veins.

Fatty degeneration of the heart is by far the most common morbid condition which leads to rupture, and many of the cases on record terminated fatally in this way. Thus in sixty-eight

examples of fatty disease, tabulated by Dr. Quain,\* rupture occurred in twenty-five; in nineteen of these the rupture was complete, and in six partial. "The actual friability of the heart appears to be quite sufficient, Dr. Quain thinks, to account for rupture in any case in which fatty degeneration is found.

Dilatation with attenuation of the ventricles has terminated in a few instances in rupture, in individuals advanced in life, but it was probably accompanied by some alteration of texture. Rupture is a very rare termination of valvular disease, and has only been observed in obstructive lesions at the aortic orifice, and where the muscular tissue of the heart had undergone some change. Cardiac aneurism, we have already said, terminates occasionally in rupture.

Abscesses or ulceration of the parietes of the heart are occasional, but rare causes of rupture; the latter by leading to cardiac aneurism, may eventuate in death by rupture. A varicose condition of one of the coronary veins may end in rupture of its coats, and death from effusion of blood into the pericardial sac, of which Albers† gives two cases; both patients had been the subject of asthma. Rupture of a coronary vein may, however, occur independent of a varicose state of this vein, of which several examples are on record; and, in cases of softening of the heart, partial rupture of the parietes may extend into a coronary vein, and so give rise to fatal hæmorrhage into the pericardial sac.

#### CAUSES OF IDIOPATHIC RUPTURE.

Rupture of the heart, being but one of the modes, in which the diseased states, of which it is a sequel, terminates, whatever predisposes to the one, may be regarded as a predisposing cause of the other. In some instances the patient had suffered great mental distress, or been a prey to profound grief. Others, from having been of active habits, had, from the effects of an injury, or other cause, been obliged to adopt a sedentary life; or had their health deteriorated from the confinement consequent upon an accident. Some had become corpulent and sedentary; in a case given by Morgagni old ulcers of the legs had been healed; in one given by Dr. Stroud, which is an example of rupture at an earlier age than usual, the patient, in addition to much mental

\* Med. Chir. Trans. vol. xxxiii.

† Brit. and For. Med. Rev. vol. xx.

anxiety, had been subject to epistaxis, which had ceased; and in a case given by M. Blaud, the patient who was fifty-eight years of age, is stated to have been much addicted to venery. Others had been the subject of various chronic pulmonary affections; while in others, again, nothing in the previous history of the case pointed to any predisposing cause.

The immediate exciting cause of the rupture has been, in some instances, a violent muscular exertion, as lifting or carrying a heavy load; this is said even to have determined rupture of the ventricle in a healthy heart. A case of the kind is mentioned by Dr. Todd,\* it was that of a brewer's drayman, who in attempting to raise a butt of porter, fell dead: on examination, a large laceration was found in the left ventricle, which, to all appearance was healthy, but it is not stated that the heart was examined microscopically, and brewers' draymen are proverbially in the habit of consuming large quantities of malt liquor, which has a remarkable tendency to induce unhealthy deposit of fat. Violent muscular exertions have occasionally, as has been said in treating of valvular disease, occasioned partial rupture, as of a fleshy column, or tendinous cord of a valve; but, I believe complete rupture never occurs in a perfectly sound heart, except as the result of external violence.

In other instances, the immediate cause of the rupture was strong mental emotion, as a fit of anger, excess of joy or sorrow, &c. It has occurred also in running quickly, in struggling, in jumping into a cold bath, in fits of retching, vomiting, or coughing, in the epileptic paroxysm, in straining at stool, en exerçant le coït, and in the slight exertion of rising in bed, or of getting into bed. Finally, it has occasionally happened when the individual was at perfect rest, and either seated in a chair, or recumbent in bed.

It has been a question with pathologists whether rupture of the ventricles occurs during their systole or diastole. The majority have argued that, because rupture of muscles occurs during a state of violent contraction, rupture of the muscular tissue of the heart must take place likewise during its systole. But there is little analogy, as Pigeaux† has shown, between the two cases: the muscles most frequently ruptured are long, and slender, are

\* Cyclop. of Anat. and Phys. vol. ii.

† Mal. du Cœur, tom. i.

attached to bony points at each extremity, and the rupture is always transverse; in the heart on the contrary, the rupture is generally parallel with the muscular fibres; its muscular tissue, besides, is short and thick, and its fibres are not attached to unyielding points.

We have seen that spontaneous rupture is almost invariably preceded by some alteration of texture, owing to which the parietes of the heart are weakened or its cohesive power is diminished. If in such a state of the organ, mental agitation, or bodily exertion lead to over-distension of its chambers, and if the cavity can not relieve itself by the natural outlet, the pressure from within outwards may become so considerable that the parietes yield at their weakest point, and give way. It is probable, therefore, as Pigeaux supposes, that the rupture occurs during the diastole, rather than the systole of the ventricles.

Why spontaneous rupture occurs more frequently in the left, than the right ventricle, is easily explained; for while the tricuspid valve readily permits a reflux of blood into the right auricle, and so relieves itself of over-distension, the mitral valve, from its unyielding nature, prevents regurgitation—hence over-distension of its cavity is not so easily relieved, and hence, rupture is more frequent in it.

#### SYMPTOMS OF RUPTURE.

Cases of this accident, so far as relates to the symptoms, may be arranged in two groups.

In one, the rupture is instantly fatal.

In the other, the patient survives the first symptoms of rupture for several hours, or days.

In either case, the rupture may or may not be preceded by certain premonitory symptoms.

When the rupture is instantly fatal, the subject of it suddenly falls to the ground, deprived of sensation and motion, sometimes uttering a cry as in epilepsy, or a moan, or exclamation; the face extremely pallid, the pulse and impulse of the heart are imperceptible, and the limbs are relaxed. In such cases, the rupture is always complete, and the rent is usually of some size. The pericardial sac is instantly filled and distended with blood, the heart compressed, and its movements are suspended.

The immediate cause of the sudden death in this accident is

evidently the compression which the heart suffers; not, as supposed by some, the loss of blood. Thus, if rupture occurs in the left ventricle, its contents instantly escape into the pericardium, which becomes distended, the auricles, and the origin of the large vessels are compressed; the left ventricle being empty, can neither transmit blood to the brain, nor to the tissue of the heart, and the coronary veins cannot return their blood; the heart is paralysed, its movements are necessarily brought to a stand, and life ceases.

When the rupture is *not* immediately fatal, the patient is suddenly seized with pain, sometimes excruciating, referred to the præcordial region, and extending to the left shoulder and arm, or to the sternum and left scapula, to the epigastrium and sternum, or to the epigastric region alone; sometimes the pain has been referred to the entire chest, and extended subsequently to both arms. It is accompanied by a sensation of constriction, weight, or burning heat, with intense anxiety, oppression, weakness, a sense of sinking, or of approaching death. The surface becomes pale and cold, and profuse perspiration sometimes breaks out over the upper part of the body. The pulse is small and feeble, sometimes scarcely to be felt, at others intermittent or slow; but it may not be altered if the septum is the seat of the rupture. In some instances, vomiting or straining, giddiness, convulsions, or spasmodic contractions of the extremities, colicky pains, or apoplectic symptoms have been present. When the pain extends from the præcordial region to the left shoulder and arm, it resembles in some respects a severe attack of angina pectoris; when it is referred especially to the epigastrium, and is attended by vomiting, and straining, it has been mistaken for the passage of a gall-stone.

The symptoms sometimes remit or subside for a short period, and then recur with increased intensity; and this may happen more than once. In other instances, they go on increasing in intensity from the first moment, until the death of the patient; which may take place within a few hours, or not for two or three days.

The interval which elapses between the first symptom of rupture and the death of the patient, varies according to the size of the rent, its situation, and course, and whether it was in the first instance partial, implicating only the internal layers of fibres, or complete. In some of the recorded cases, a coagulum had closed

the orifice, and prevented for a time the escape of blood. In others, the rupture was oblique, forming a sinuous passage for the blood, by which the fatal result was retarded. In one recorded by Dr. Townsend,\* the rent, which was half an inch in length, was closed from the inside by one of the *carneæ columnæ*, which had been torn across, and projected through the orifice; in this case, however, death was instantaneous. When the septum of the ventricles is the seat of the rupture, death is seldom instantaneous, and the patient may live for several days. In a case of this kind recorded by Dr. Latham, a loud systolic murmur, in the præcordial region, was for the first time audible some days after the first symptoms of rupture. In a case recorded by Rostan, a spontaneous cure is said to have taken place by the cicatrization of the rent.

In some of the cases on record, the patient, previous to the first symptoms of rupture, had been in the enjoyment of excellent health; and, had never complained of any symptom referred to the heart. In others, premonitory symptoms of some kind had been present for a longer or shorter period, many of which are common to various organic diseases of the heart. Thus, in several instances, the patient is said merely to have felt unwell for some days previously, or to have complained of a slight sensation of uneasiness in the præcordial region. In others, the symptoms were more marked, and were referred sometimes to the heart, sometimes to the lungs or throat; at others, rather to the abdomen or head. The principal of these were palpitation and pain in the præcordial region; cough, dyspnoea on slight exertion, the sensation of a load in the chest, a sense of choking referred to the throat; epigastric pain, nausea, various dyspeptic symptoms, and lumbar pains, which were supposed to be nephritic; vertigo, faintness, syncope, apoplectic symptoms; malaise, debility, sensations of sinking, giddiness, and profuse perspiration on slight exertion.

#### DIAGNOSIS OF RUPTURE OF THE HEART.

The accidents with which rupture of the heart is most liable to be confounded are, rupture of the diaphragm, rupture of the aorta, or of an aneurism of this vessel, and apoplexy. Rupture of the diaphragm may be as quickly fatal as rupture of the heart, and like it may be the result of a fall from a height, or of a heavy vehicle

\* Cyclopædia of Medicine, vol. iv.

passing over the body, or of some sudden, violent muscular exertion; or it might occur during violent vomiting, or straining. But here the analogy ceases—in rupture of the diaphragm, the pulse and impulse of the heart are perceptible; the accident is accompanied by nausea, or vomiting, with risus sardonicus, inability to move, and a greater amount of dyspnœa; while, if the rupture was sufficiently large to permit some of the contents of the abdomen to pass into the thorax, there would be, in addition, flattening of the former cavity, and dilatation of the latter.

Rupture of the aorta, or of an aneurism of the ascending portion of the arch into the pericardial sac, in general proves as quickly fatal as rupture of the heart, and could not be distinguished without a post-mortem examination. Rupture of an aneurism of any other part of the aorta might, also, be mistaken for it, but is seldom so immediately fatal; while, the previous history, and the symptoms which immediately follow the rupture, are different in the two cases.

One form of apoplexy, in which the death is almost instantaneous, might be mistaken for rupture of the heart; but, if the patient lives for a few hours, the labouring pulse, stertorous breathing, congested countenance, and the presence of paralysis will always enable us to distinguish between them.



## CHAPTER XXI.

INCREASED ACTION OF THE HEART WITH ENLARGEMENT OF THE THYROID GLAND, AND PROMINENCE OF THE EYE-BALLS—ANATOMICAL CHARACTERS—SYMPTOMS.—CAUSES—DURATION AND TERMINATION OF—TREATMENT.

BEFORE proceeding to the consideration of the *Inorganic* affections of the heart, a peculiar combination of symptoms requires notice, which appears to partake partly of the nature of functional derangement, and partly of organic disease, or to be an intermediate link between them. This morbid condition commences always in functional derangement of the heart; after persisting as such for an indefinite period, it either subsides under treatment, or it passes into organic disease of the organ.

One of the first to notice this morbid condition was Dr. Parry,\* Dr. Graves† recalled attention to it, and we are indebted to Sir Henry Marsh‡ for the first record of the pathological appearances in fatal cases. Since then, additional cases have been placed on record by Drs. Macdonnell,§ Begbie,|| Cooper,¶ Stokes,\*\* Banks,†† Taylor,‡‡ Romberg, Henoch, &c.

## ANATOMICAL CHARACTERS.

The pathological appearances observed in the few fatal cases on record were presented by the heart, by the thyroid gland, and vessels of the neck, and by some other organs. Those presented by the *heart* were pretty uniform, and such as might be expected to follow long continued derangement. In all, the heart was enlarged, the enlargement being due to dilatation of the chambers, with generally some hypertrophy of the parietes. All the

\* Collections from unpublished writings, vol. ii. || Ed. Month. Jour. Feb. 1849.

† Clinical Lectures, 1835.

¶ The Lancet, May, 1849.

‡ Dub. Jour. of Med. vol. xx. 1842.

\*\* Treat. on Dis. of the Heart, 1854.

§ Ibid. vol. xxvii. 1845.

†† Dub. Hosp. Gaz. 1855.

‡‡ Med. Times and Gaz. May, 1856.

chambers were dilated; or it predominated in the auricles; in one, the auriculo-ventricular valves, particularly on the right side were thickened at their margin, from deposit under the endocardium; in another the auriculo-ventricular orifices were dilated. The inferior cava was unusually large, and the aorta small compared with the pulmonary artery in one case, and in one the muscular tissue of the heart was soft and flaccid.

The morbid condition of the *thyroid gland* consisted sometimes in simple hyperæmia, or hypertrophy, in others the enlargement of the gland was combined with the development of cysts in its interior; and in some it presented the characters of vascular or aneurismal bronchocele. The gland after death was always smaller than it had been during life, and the enlargement was either uniform, or it predominated in one lobe; the surface was lobulated or smooth, its consistence greater than natural, and a section of it exhibited occasionally cysts containing either a clear fluid, or a yellow liquid like honey, or a dark fluid like coagulated blood. The thyroid veins in one case were remarkably dilated, in another the thyroid arteries were enlarged and tortuous. The internal jugular veins were generally much dilated, sometimes this increase in size was greatest on the right, sometimes on the left side.

The morbid appearances in other organs were such as are generally consecutive to organic disease of the heart, and had nothing peculiar in them. They were, congestion of the lungs, anasarca, and serous effusion into the pleural or peritoneal cavities, enlargement and congestion of the spleen, the liver presenting the nutmeg character, or in the first stage of cirrhosis; kidneys large and soft, or in the early stage of Bright's disease.

#### SYMPTOMS.

The prominent and characteristic features of this morbid condition, when fully developed, are—

1. Violent and long-continued palpitation.
2. Enlargement of the thyroid gland.
3. Prominence of the eyes.
4. Visible pulsation of the large arteries of the neck.
5. Increased quickness of the pulse.

The order in which these symptoms make their appearance is usually as follows; commencing with palpitation, enlargement of

the thyroid gland with increased action of the arteries of the neck follows, the pulse becomes more rapid, and after a period which varies in different cases, the eyes are observed to be more prominent than natural. Although this is the usual sequence of the symptoms, enlargement of the thyroid has occasionally set in first, and been followed by the others. Sometimes prominence of the eyes follows palpitation, and the thyroid gland does not begin to enlarge until some time subsequently, occasionally extreme rapidity of the pulse was the first symptom noticed; and prominence of the eyes may never occur, although all the other symptoms are well marked.

*Palpitation* seems to be the starting point in these cases; the action of the heart is stronger than natural, and more diffused, sometimes visible over the whole præcordial region, it is rapid but usually regular, and the palpitation is distressing to the patient, and constant, though increasing under excitement. It is occasionally accompanied by pain in the left side, or by noises in the head, which have sometimes been so loud as to prevent sleep. In a case mentioned by Dr. Graves, the first sound of the heart was so loud during the paroxysms as to be audible at some distance from the patient. The region of the heart's superficial dulness is somewhat creased; but unless the heart has become enlarged, this is not very perceptible; the impulse is not forcible or heaving, unless the ventricles have become hypertrophied; or irregular, unless dilatation of their chambers has supervened.

In some of the cases, a bruit de soufflet was audible in the præcordial region, in the majority there was none; when present, it accompanied the first sound of the heart, and was loudest over the aortic valves, from which it extended a short distance in the course of the aorta; in one instance, it was attended by fremitus in this situation; in another, in which however organic disease had set in, a triple sound was audible in the præcordial region.

*The enlargement of the thyroid gland* is sometimes uniform, sometimes it predominates in one lobe; the gland may attain a large size, from two to four times its normal dimensions, forming a considerable tumor, or the swelling may be inconsiderable; the gland varies in size in the same patient, increasing under excitement of the heart, or in paroxysms of coughing, and diminishing as the heart's action becomes tranquil. The swelling in general arises

gradually and imperceptibly; in a very few cases it set in suddenly, in one of these it was noticed soon after a violent fit of retching or vomiting. The surface of the tumor is generally smooth and uniform, sometimes soft and elastic; in the advanced stage it becomes firmer, harder and diminished in size. A strong pulsation is felt over it; a purring thrill is sometimes communicated to the hand, and the thyroid arteries are occasionally felt to be enlarged. The carotid arteries pulsate strongly, and the patient complains of a sensation of beating or fulness in the neck; or where the tumor is larger, of a feeling of constriction, or tightness in this part. On applying the stethoscope a loud continuous venous murmur is heard in the neck, which is sometimes musical; in a case given by Dr. Banks, the pulsation diminished in the recumbent posture, and the murmur became almost inaudible. This enlargement of the thyroid differs from ordinary bronchocele, as remarked by Dr. Graves, "in not attaining a size at all equal to that observed in the latter," and "in remaining stationary just at that period of its development when the growth of the latter usually begins to be accelerated."

The pulsation in the thyroid gland is in part communicated by the carotid arteries, assisted perhaps by the enlargement of its own arteries; the gland when it enlarges laterally, overlaps the common carotid on each side, and if these vessels pulsate strongly an impulse may be communicated to the gland, which is perceptible to both the eye and hand; indeed when enlargement of the thyroid is limited to one side, it may simulate carotid aneurism; on desiring the patient to make the effort to swallow, however, it will be seen to rise with the larynx. In other cases the arteries and veins of the gland are both enlarged, a fremitus is communicated to the hand, and the swelling presents some of the characters of aneurism by anastomosis.

Enlargement of the thyroid is almost always preceded by increased action of the heart, and of the carotids; it usually precedes the prominence of the eyes, though in a few instances it has been the last symptom noticed. Palpitation and enlargement of the thyroid often occur independent of any prominence of the eyes; or palpitation, and prominence of the eyes may occur without any increase in the size of the thyroid gland.

*Prominence of the eyes.* The most characteristic feature of the

fully advanced stage of this affection is a prominence of the eyeballs, giving the eyes a wild and staring appearance, which is sometimes so considerable that the lids cannot be closed, and in deep sleep the globe of the eye is partially uncovered; yet it is remarkable that in no instance has injury to vision followed. In the majority of cases, this is a late phenomenon. It is always preceded by palpitation, by increased action of the arteries of the neck, and most always by enlargement of the thyroid gland. Dr. Graves has, however, recorded a case where it preceded the latter by some months. In some instances it has quickly followed the enlargement of the thyroid, in others it has come on at a much later period; or it may never ensue, although all the other symptoms are well-marked. In general, it supervenes slowly and gradually; in a few instances, it sets in suddenly, either after a violent fit of coughing, or retching, or without any apparent exciting cause; as in a case given by Mr. R. Taylor, where the patient having gone to bed well, on awakening could scarcely close the eyelids.

*Pulse.* The pulse in this affection is always more rapid than natural, often considerably so; sometimes this was the first symptom noticed, and it preceded the palpitation; it is regular as long as the derangement of the heart's action is purely functional; when the left ventricle becomes dilated, the pulse presents the ordinary characters of dilatation, and is weak, intermittent, or irregular, as well as rapid. The frequency of the pulse ranges between 110 and 140; it is seldom below the former, though it often rises above the latter. Thus in a case given by Dr. Macdonnell, it was never under 120, and sometimes as high as 200; in one given by Dr. Banks, it was so rapid as to be impossible to count; and in four cases given by Mr. Taylor it ranged between 134 and 144.

*Sex—Age.* This affection is much more frequent in the female than the male; out of twenty-two cases, of which accurate records have been given in British journals, nineteen were females, and three only males. It is observed in both married and unmarried females, and in those who have borne children, equally as in those who were never pregnant. Sir H. Marsh says he has generally observed it in tall persons, but some of the subjects of the foregoing cases are stated to have been low in stature. It is most common

between twenty and thirty years of age ; the youngest subject in the cases above alluded to was seventeen years of age, the oldest fifty-five ; both these were males.

#### CAUSES.

This morbid condition is generally preceded either by some exhausting discharge, or by something which equally deranges the general health, lowers vital power, and deteriorates and impoverishes the blood. Thus, in some instances the patients had suffered repeated losses of blood either from the uterus, hæmorrhoidal vessels, or by epistaxis, in one from an accidental wound ; others had long laboured under profuse leucorrhœa, or diarrhœa ; in some, the affection had been preceded by hysterical symptoms or epilepsy ; while mental anxiety, prolonged lactation, insufficient food, or over-work had impaired the health of others.

The ultimate result of these several causes of deterioration and impoverishment of the blood is, to induce an anæmic state of the system ; the affection may therefore be regarded as one of the rarer results of *Anæmia*, as first pointed out by Dr. Begbie. Indeed the subjects of it present the ordinary characters of anæmia ; they are generally pale and chlorotic looking, and often labour under amenorrhœa, leucorrhœa, or menorrhagia ; they suffer from indigestion, impaired appetite, disturbed sleep, short cough, coldness of the extremities, headache, ringing in the ears, and palpitation ; while various nervous or hysterical symptoms, as intercostal neuralgia, or spinal irritation, are occasionally present.

The palpitation, the increased action of the arteries of the neck, and the rapid pulse are common to this affection and anæmia, and depend apparently upon the same cause. The enlargement of the thyroid gland, and the prominence of the eyes are involved in more obscurity.

*Cause of enlargement of the Thyroid.*—Sex predisposes to enlargement of this gland, it being naturally larger in the female than the male, as noticed nearly two hundred years ago by Wharton.\* “It contributes much (he quaintly observes) to the rotundity and beauty of the neck, filling up the vacant spaces round the larynx, and making its protuberant parts almost to subside, and become smooth, particularly in females, to whom for this

\* Adenographia, 1659.

reason a larger gland has been assigned, which renders their necks more even and beautiful."

Slight enlargement of the thyroid is not at all uncommon in æmic females, independent of any prominence of the eyes, as well as in certain cases of organic disease of the heart, and the enlargement of the gland is preceded by increased action of the heart, and of the large vessels of the neck; indeed Dr. Parry,\* on witnessing this sequence, suggested as a probable use of the thyroid, that it served as a *diverticulum* to the cerebral circulation. "I have so often seen (he says) swelling of the thyroid gland follow diseases of the heart, and other maladies in which the blood propelled with excessive momentum to the vessels of the head, and yet at the same time have observed such sudden augmentations and diminutions of the swelling, that I have suspected the gland itself to be intended as a diverticulum for blood disposed to flow with too great force to that important organ, the brain." A nearly similar view has been recently advocated by Mr. Simon; he considers "that the thyroid gland receives under certain circumstances a large share of the blood which would otherwise have been applied to the brain;" "the thyroid arteries arising close to the cerebral, and being large, and also very regular."

This view is supported by the fact, that the gland increases in size when the palpitation is violent, and the swelling diminishes as the action of the heart becomes tranquil; while in its advanced stage, if dilatation of the left chambers of the heart supervenes, the blood being no longer transmitted with increased force, or in increased amount, the thyroid gland not only ceases to enlarge, but visibly shrinks.

#### CAUSE OF PROMINENCE OF THE EYES.

This remarkable phenomenon is usually attributed either,

1. To protrusion forward of the eyeballs by abnormal deposit at the back of the orbit; or,
2. To actual enlargement of the globe of the eyes; in other words to hydrophthalmia.

Those who refer it to the former suppose its cause to lie either,

1. In œdema of the cellular tissue at the back of the orbit,
2. In venous congestion of the tissue in the same part,
3. In increased deposition of fat at the back of the orbit.

\* General Pathology, vol. i, p. 152.



Each of these theories is insufficient to account for its appearance in both eyes at the same time, and to the same extent; indeed if it depended upon either of them, the eyes would be often protruded laterally, and divergent or convergent strabismus would result; while the eyeballs could not be restored to their normal position by gentle pressure upon the lids, as Mr. White Cooper\* has shown can be done.

The theory that the eye-balls are enlarged is advocated by Dr. Begbie† and Stokes.‡ “We cannot but conclude (Dr. Stokes says) that the enlargement is owing to an actual increase in the vitreous and aqueous humours of the eye,” “that it is an example of double hydrophthalmia.” Dr. Begbie supposes the increase to be in the chamber of the vitreous humour alone. But there is no enlargement of the globes of the eye in this affection; hydrophthalmia never occurs simultaneously in both eyes, and to the same extent; or without alteration of texture, and impairment of vision; indeed, in general hydrophthalmia, the shape of the eye is altered, the eyeballs are more or less deformed, and the cornea is partially or generally opaque; whereas in this affection, the iris and cornea are natural, the pupil is regular, the sclerotic of the natural colour, and the sight unimpaired.

Mr. Dalrymple's theory, that it depends in part upon “absence of the proper tonicity of the muscles by which the eyes are retained in their natural position in the orbits” is the only one free from objection; but it throws no light upon the *cause* of loss of tonicity in these muscles, which must, in my opinion, be sought for in the nervous system, and particularly in the nerves which supply the recti muscles of the eye. Thus, the third pair of nerves which supply motor power to three of the four recti muscles, are in contact in the cranium with the cavernous sinuses, and lie between the posterior cerebral and superior cerebellar arteries; we can easily understand, therefore, that if the increased action, observed in the arteries which come off from the arch of the aorta, extends to their branches within the cranium, or if the current is impeded in the sinuses at the base of the brain, these nerves may suffer compression, the effect of which would be to diminish the tonicity of the muscles supplied by them; the globes of the eyes then not

\* The Lancet, May, 1849.

‡ On diseases of the Heart, p. 295.

† Ed. Month Jour. Feb. 1849.



being fully supported, protrusion would follow as a matter of course. Why this does not occur more frequently, probably depends upon the thyroid gland acting as a diverticulum to the blood which would otherwise be transmitted to the brain; when it no longer acts as such, the eyes become prominent.

It might be objected that only three of the recti muscles are supplied by the third nerve; but we know that in the operation for strabismus, the division of the internal rectus alone is occasionally followed by prominence, and a staring appearance of the eye, with sometimes divergent strabismus; and if the latter is attempted to be remedied by division of the external rectus, the eye becomes still more staring and prominent.

#### DURATION AND TERMINATION.

This affection is essentially chronic in its progress, extending always over many months, sometimes over years. Thus, in some instances its duration was between six and seven years, in the majority it was between two and four. The duration of the several symptoms is not the same, the enlargement of the thyroid gland being always the last to subside, and sometimes never altogether disappearing; while the palpitation, quick pulse, and prominence of the eyes subside, and finally disappear as the general health improves.

The termination in the great majority of cases is in a return to health; out of twenty-two cases referred to above, eighteen recovered more or less perfectly, and four died. In the latter, organic disease of the heart supervened, and the termination was in general dropsy, or in effusion into the serous cavities; with the usual alterations in the lungs, liver, &c., observed in fatal cases of organic disease of the heart.

#### TREATMENT.

A strong argument in favour of the anæmic origin of this affection is, that the treatment found most effectual in anæmic cases generally, proves equally so in it; and that antiphlogistic, and other measures, which prove hurtful, or injurious in the former are no less so in it. The causes which have produced the anæmia must be ascertained if possible, and as this is often some exhausting discharge, our measures in the first instance must be directed to the relief or removal of it.

The medicines upon which we rely especially in this affection are the preparations of iron; and those found most effectual in ordinary cases of anæmia prove generally equally so in it. The muriated and acetated tinctures, the mixtures of the pharmacopœia, the subcarbonate, sulphate, tartrate, iodide, ammonio-citrate, or the citrate of iron and quinine, may each in their turn be employed; and, as they generally require to be continued for some time, the dose, the form, and the combination should be so regulated as not to disorder the stomach; and it is generally better, instead of persevering in the use of any one, to vary the form occasionally. The muriated tincture of iron with bicarbonate of soda in the state of effervescence, as originally used by the late Mr. Carmichael, is favourably spoken of by Mr. White Cooper; it has agreed, he says, where iron in other forms has disagreed: another preparation which he recommends is the liquor oxysulphatis ferri, in the dose of from three to twelve drops. The local application which has generally proved most effectual in quieting the inordinate action of the heart, is a belladonna plaster to the præcordial region.

All lowering treatment as a general rule is to be avoided; when from mistaken views respecting the nature of this affection, leeches, bleeding, and low diet were employed, they invariably added to the patient's distress, and prolonged the period of convalescence. Mr. W. Cooper states, that in two instances patients who subsequently came under his care, had been subjected to a mercurial course for the supposed hydrophthalmia, and much mischief was the result.

“The progress towards recovery is generally (as Mr. Taylor\* remarks) slow, and the treatment may require to be prolonged over many months; but if judiciously selected, and carefully persevered in, we may look forward with confidence to the restoration of the general health, and the complete disappearance of the deformity caused by the unnatural prominence of the eyes.” The swelling of the thyroid gland seldom entirely disappears; but as “the enlargement is rarely to any extent, and as a certain amount of diminution invariably takes place, the persistence of a slight and scarcely perceptible fulness is not a matter of any importance.”

\* Med. Times and Gaz. May, 1856.

## CHAPTER XXII.

**INORGANIC AFFECTIONS OF THE HEART.—CLASSIFICATION OF.—FUNCTIONAL DERANGEMENT OF THE HEART DEPENDING UPON ANÆMIA, PLETHORA, OR CARDIAC CONGESTION; IN NERVOUS AND HYSTERICAL SUBJECTS; IN DYSPEPSIA, GOUT, AND DEBILITY; IN DEFORMITY OF THE CHEST, AND IN CONGENITAL SMALLNESS OF THE HEART.—SYMPTOMS, CAUSES, AND TREATMENT.**

THE abnormal conditions which we have now to consider are characterized, as a general rule, by deranged or disordered action of the heart. They are of frequent occurrence, they are met with in a variety of forms, and they derive interest from the circumstance that the combination of symptoms they sometimes present, is very similar to that of organic disease; indeed, so close is the resemblance, that the diagnosis at times is one of some difficulty. They are also frequently as distressing, or even more so, to the patient; their course and progress are, however, different; and they yield to treatment with greater facility.

In describing the symptoms of organic disease, we found that palpitation, tumultuous or irregular action of the heart, intermission and irregularity of the pulse, dyspnœa, cough, pain in the præcordial region, or in the left side, visible pulsation in the large arteries of the neck, and a bruit de soufflet on auscultation, are ordinary symptoms. Any one of them may, however, be present, when no disease of the valves, or orifices, of the parietes, or investing membrane of the heart exists; and it is a matter of some moment to be able to distinguish between them; particularly as the treatment adapted to the one would be very likely to prove injurious in the other.

Disordered action of the heart (independent of organic disease of the organ) is met with—

1st. Where the blood has undergone changes in its quality,

quantity, or distribution. Under this head, palpitation from *anæmia*, *plethora*, and cardiac congestion may be included.

2nd. Where some disordered condition of the nervous system exists; which includes the nervous and hysterical forms of palpitation.

3rd. In derangement of function of other organs, or in some general disorder of the system, which will include palpitation from *dyspepsia*, *gout*, and debility.

4th. When a disproportion exists between the capacity of the thorax and the size of the heart—as in contraction, and deformity of the chest from lateral, or angular curvature, or other causes—or where the heart is congenitally smaller than natural.

Derangement of the heart's action, depending upon any of those conditions, is more likely to be mistaken for organic disease than the latter for the former; indeed, the number of persons who seek advice, under the impression that they labour under disease of the heart, is perhaps as great as of those actually suffering from it. This arises partly from the inconvenience the patient suffers being greater, the heart's action being more readily excited, and his attention being more constantly directed to it. In fact, organic disease, unless somewhat advanced, or unless symptoms of obstruction to the circulation have set in, may not cause much inconvenience, and many of the individuals who seek relief at hospital will not even allow that they suffer from palpitation, although when we come to examine the heart we find its impulse much increased, or indications of disease of the valves present.

Functional derangement of the heart, depending upon alterations in the quality, quantity, or distribution of the blood, may have its cause—

1. In deficiency in one of the most important constituents of the blood (the red corpuscles), of which *anæmia* is the type.

2. In excess of blood in the system at large, of which general *plethora* is an example.

3. In local excess of blood, or local *plethora*, as in *cardiac congestion*.

*Anæmic Palpitation*.—The state denominated *anæmia*, or *spanæmia*, from *σπавος*, poor, and *αἷμα*, blood, includes all those cases in which the blood is impoverished, and its quality deteriorated. In this state, the solid constituents of the blood are diminished,

and the water is increased; its density consequently is less than that of healthy blood. All the solid constituents do not, however, undergo equal diminution: this is limited in a great measure to the red corpuscles. Thus, according to M. Becquerel,\* 1000 parts of healthy blood contain:

Water	...	...	...	from 770 to 830 parts
Red corpuscles	...	...	...	from 120 to 140 „
Fibrin	...	...	...	from 2 to 3 „

In anæmia, the blood, according to him, contains in 1000 parts:

Water	...	...	...	from 830 to 880 parts
Red corpuscles	...	...	...	from 40 to 120 „
Fibrin	...	...	...	from 2 to 3 „

“The red corpuscles in this affection appear also to contain less hæmotosin, but the fibrin is not perceptibly altered. The albumen of the serum generally maintains its normal proportions except in extreme cases, when it falls from 70 or 80 parts in 1000, to 65.”

M. Becquerel makes three degrees of anæmia founded upon the proportion of the red corpuscles.

In the first degree (slight anæmia) the red corpuscles are between 100 and 120 in 1000 parts.

In the second degree (decided anæmia) the red corpuscles are between 80 and 100 in 1000 parts.

In the third degree (aggravated anæmia) the red corpuscles are below 80.

Anæmia is generally a consecutive state, though it also occurs as an idiopathic affection; and it may be brought about in a variety of ways. Thus, it may be directly the result of excessive loss of blood, or it may be more slowly produced by exhausting discharges, or the escape of the solid constituents of the blood with the excretions, as in profuse suppuration, protracted leucorrhœa, prolonged lactation, diarrhœa, diabetes, Bright's disease, &c. It may be also the result of insufficient or innutritious food, or unhealthy habitation, or locality; or of previous disease, acute or chronic, or it may be produced by the remedies employed for their relief, as bleeding, low diet, purgatives, and mercury. The

\* Gazette des Hôpitaux.

diseases upon which it is most liable to supervene, are functional disorder and organic disease of the uterus, chronic diseases of the heart, spleen, and stomach, acute rheumatism, and acute inflammatory affections generally, particularly when very actively treated; chronic bronchitis, intermittent fever, secondary syphilis, and cancer.

When this morbid condition is marked, it is characterised by an exsanguine condition of the surface, and a yellowish, sallow, or waxy hue of the skin; the face is sometimes bloated, at others there is a dark circle round the eyes; the lips and gums are colourless, the tongue is pale, the appetite capricious, usually bad; and nausea, vomiting of food, pyrosis, cardialgia, flatulence or constipation are present. Headache, limited often to one side of the head, or giddiness or vertigo are complained of by some; by others, ringing, buzzing, or whizzing noises in the ears are experienced, particularly when the anæmia is due to profuse hæmorrhage. Palpitation, and breathlessness on slight exertion, such as ascending stairs, are very generally experienced, and the patient often complains of pain in the side, particularly the left. Languor, weakness, disinclination to bodily exertion, fainting, or a tendency to it, and hysterical fits are occasionally present. The functions of the skin are imperfectly performed; and, finally, the catamenia are either absent, scanty, or too profuse.

The cardiac phenomena are likewise very characteristic; palpitation is invariably present, and it is increased by very trifling exertions, it is usually referred to the præcordial region, and is often accompanied by a sensation of fluttering in the epigastrium; the action of the heart is always more rapid than in health, and there is often visible pulsation in the arteries of the neck; a bruit de soufflet is audible sometimes at the base of the heart, generally in the large arteries which come off from the arch of the aorta, and a continuous murmur is almost invariably heard in the veins at the root of the neck.

*The noises in the head*, the sounds of beating, blowing, singing, or whizzing, seem to be due to increased friction between the blood and the lining membrane of the arteries of the brain, particularly in those which pass through bony canals, which are incapable of adapting themselves to the varying amount of blood they receive at different periods. This symptom is usually more complained of

when the anæmia is the result of profuse hæmorrhage, than when it is associated with chlorosis; in the former also, it is often increased by the recumbent posture, and the ringing sound is sometimes so loud, when the patient lays her head on the pillow, as to prevent sleep. It is compared to the ringing of bells, the noise of bellows, &c.; a patient under my care compared it to the hissing sound produced by rapidly whirling round a switch. Micrania when present, is rather a symptom of hysteria, in which anæmia is not unfrequently associated. Vertigo is occasionally complained of, but it is more common in the plethoric form.

*The Pains in the side*, particularly the left, though sometimes due to neuralgia, are frequently the result of flatulent distension of the stomach or colon, the result of dyspepsia, or of the excessive secretion of gas by the gastro-intestinal mucous membrane, so common in hysterical cases. These pains are always increased by leeches or cupping, and are relieved by treatment which improves the digestion and removes anæmia.

*The frequency of the pulse* appears to be due to the thin and watery state of the blood, the heart's action being quickened in order, by its frequency, to compensate for the poverty of this fluid. The muscular tissue of the heart, likewise, being imperfectly nourished, its action is more readily excited than in health, while the weakness of its parietes prevents the cavities from being completely emptied; less blood is consequently transmitted at each ventricular systole, and the large vessels which come off from the arch of the aorta are imperfectly filled—hence the pulse is compressible, and jerking as well as quick, resembling in this respect the pulse of aortic regurgitation. Change of posture has a more remarkable influence upon the pulse, and the difference between the number of beats in the recumbent and erect posture is greater than in health.

*Visible pulsation in the arteries of the neck* appears to be due to the unfilled state of these vessels, and is often combined with a murmur and fremitus in them. The patient sometimes complains more of the sensation of beating in the neck than of the palpitation, particularly if any enlargement of the thyroid gland is associated with the anæmia.

*Physical Signs.*—The physical signs of anæmia are furnished

partly by percussion and palpation, but principally by auscultation. Those furnished by percussion and palpation are rather negative than positive; thus, the extent and amount of dulness on percussion in the præcordial region, does not exceed that of health, and the impulse is circumscribed, sharp, and knocking, or occasionally thumping, but never prolonged or heaving.

On *auscultation* the sounds of the heart are observed to succeed each other rapidly, and the interval of repose is shortened; both sounds are short and they may be either loud and clear, giving a ringing sensation to the ear, or loud and abrupt. If the heart's action is very rapid, the first sound is sometimes as short as the second, and the second sound is then occasionally less distinct than natural.

*The abnormal sounds* which characterize anæmia have their seat at the aortic orifice, in the large arteries, and in the veins of the neck; at the aortic orifice and in the large arteries the abnormal sound is a bruit de soufflet; in the veins it is a continuous or a musical murmur.

*The bruit de soufflet* at the aortic orifice is a soft, blowing, seldom rough or harsh sound, it accompanies the ventricular systole, the impulse and the pulse, is loudest from the base of the heart in the direction of the current of blood from the left ventricle, and is not audible at the apex of the organ. In many instances, however, this murmur is not heard, or it is not constant, being audible only when the heart's action is excited.

*The arterial bruit de soufflet* is more frequent and more constant than the other; the arteries in which it is heard are the carotids and subclavians, it is often also audible in the abdominal aorta, the iliac, and femoral arteries. Its general character is short and whiffing, it accompanies the ventricular systole and the first sound of the heart. Sometimes a *double sound* without any murmur is heard in the vessels of the neck, and on making pressure with the stethoscope this is converted into a single hoarse murmur; occasionally a *double murmur* is audible in the same situation. The arterial bruit is audible only over the line of the artery, its character is altered by compressing the vessel with the stethoscope, when it is rendered sharper, and if a bruit was not audible it is readily developed by making pressure on the artery, a *fremissement* being at the same time often felt.



*The double sound in the arteries of the neck* depends in a great measure upon the unfilled state of these vessels. In anæmia there is a deficiency of tone in the coats of the arteries, they are relaxed, at least they have not the tension of healthy vessels; in addition, less blood is transmitted from the heart at each ventricular systole, the large arteries are consequently imperfectly filled, and at each ventricular diastole there is a reflux in the carotids which gives rise to the second sound.

*The continuous venous murmur* has its seat in the jugular veins, particularly the internal, and the veins which open into them; it is usually audible upon both sides of the neck, sometimes only on one side, it is best heard a little above the clavicles and is inaudible at the angle of the jaw. The position in which to place the patient in order to hear this murmur is either sitting or standing, with the face inclined towards the opposite shoulder, and the chin somewhat elevated so as to render tense the fascia of the neck, and the coats of the vein; if the head is inclined to the side we are examining and bent forward so as to relax these parts, the murmur will diminish or cease; it is generally inaudible also in the recumbent posture. In order to hear this murmur it is essential to employ the stethoscope; altering the pressure of the stethoscope alters its tone remarkably, diminishing or increasing its intensity, rendering it hoarse, or converting it into a musical murmur according to the degree of pressure. When stronger pressure, sufficient to obliterate the current in the vein, is made, either with the edge of the stethoscope or with the finger above it, the murmur instantly ceases.

*The musical venous murmur* is merely a variety of the continuous murmur, it is always preceded by it, and can be converted into it by increasing the pressure upon the vein with the stethoscope, it is therefore to be regarded as merely an exaggerated form of it. The mechanism by which the continuous and the musical venous murmur are produced, has been already noticed (p. 157), it will be sufficient here to observe, that these murmurs in my mind are not actually present in the veins of the neck, but they are mechanically developed by the pressure of the stethoscope; and in order that they should be heard, the coats of the vein must be rendered tense, and the fascia of the neck and the other parts covering the vessel, must be put upon the stretch.

**Plethoric Palpitation.**—Symptoms simulating cardiac disease are, occasionally, the result of an entirely opposite condition to that in anæmia. In *plethora* blood is in excess, the blood vessels are distended, and this fluid, instead of being impoverished, is richer than natural, the proportion of water being less and that of the red corpuscles greater than in health. “It has generally been supposed (Andral observes) that the fibrin of the blood is augmented in plethora; such, however, is not the case, it may amount to its maximum but never exceeds it. The quantity of albumen is not increased either; the red globules are the only element increased, they are increased to the full physiological limit, and sometimes exceed it.”

In true or sthenic plethora the functions of digestion and assimilation are active, blood is rapidly formed; the vascular system is loaded, the impulse of the heart is stronger than natural, and the pulse is full and strong; the individual is short-winded, the complexion is florid, and headache, vertigo, somnolency, with sometimes throbbing of the arteries of the neck are present. Palpitation from this cause is not often met among the labouring classes, but is not uncommon in men who previously had been of active habits and had relinquished them, but continue to eat and drink as before; as well as in sedentary females of a certain age, who, as Dr. James Johnson says, “eat a great deal too much, walk a great deal too little, and consequently have a plenitude of the vascular system.” It is liable to occur also in individuals in whom some drain upon the system had been checked, as bleeding from piles, or where an old ulcer, or an issue of long standing had been healed up; and in women about the period of the cessation of the menses.

Palpitation from *obesity* comes also under this head; here, however, there is generally deficient vital or nervous power, and the plethora is of the false or asthenic kind. Obesity may, as Dr. Hope\* observed, give rise to palpitation in more than one way, not only “by the plethora with which it is usually accompanied; but, by the weight and inelasticity of the thoracic and abdominal parietes, whence the free expansion of the ribs and descent of the diaphragm are prevented, as well as by the unusual resistance offered to the heart’s propulsive action, in consequence of the ex-

\* Cyclop. of Pract. Med. vol. iii.

tended sphere of the circulation and the pressure of the adipose tissue on the vessels."

The palpitation from which some females suffer during the early months of *pregnancy*, is in some instances due to plethora, the result of suppression of the catamenia; in others it appears rather to be connected with hysteria, or to be the result of sympathy; in the former a bleeding from the arm is sometimes effectual—in the latter, antispasmodics combined with tonics, and attention to the diet and the bowels, are the means from which most benefit will be derived. In the advanced stages of pregnancy, if palpitation is a troublesome symptom, and if combined with any dyspnœa, it generally has a mechanical cause, viz., the pressure of the gravid uterus upon the large vessels in the abdomen, and upon the diaphragm preventing its descent. In such cases, we can do little more than endeavour to obviate the effects of pressure by position and attention to the bowels.

*Palpitation in local plethora.* Local plethora, limited to the heart and lungs, the result of the arrival of the blood in excess at the right side of the heart, presents many of the cardiac phenomena observed in general plethora. Its symptoms according to Dr. Calthrop Williams\* are "accelerated breathing, dyspnœa, and dry paroxysmal cough; the feeling of dyspnœa is sometimes considerable, though the respirations are not more frequent than natural." "The impulse of the heart is always increased, laboured, and struggling, but the force of the arterial pulse is diminished because the heart is unable to circulate the blood with its accustomed energy." The most common cerebral symptoms are "vertigo, swimming in the head, ringing in the ears, temporary disorder of the intellectual functions, and a peculiar tottering of the gait, which comes on suddenly and compels the patient to grasp the nearest object for support. There is often also some pain or uneasiness about the præcordial region which the patient endeavours to relieve by deep inspirations, or a single short cough."

Among the causes of local plethora, muscular efforts beyond the patient's strength, and mental emotions of an exciting kind are the chief. "We have frequent examples of the influence which the former exercises in producing congestive palpitation (Dr. Calthrop Williams† observes), among the young members of

\* On Functional Diseases of the Heart. † Ibid.

cricket, or rowing clubs. The violent and long continued muscular efforts made by many of these young men in the prime of life and vigour, without sufficient training, or preparation of the respiratory organs, often give rise to very severe and obstinate paroxysms of palpitation, which have a tendency to recur again and again from the slightest exciting cause." In hospital practice, we meet with examples of permanent palpitation from similar causes in young persons of both sexes, servants and apprentices particularly, who have been put to work which is above their strength, or who have to walk a long distance to their daily employment, or to undergo muscular exertions beyond their powers. When these causes have been in operation for a lengthened period, permanent enlargement of the heart is generally the result.

Functional derangement of the heart depending upon disordered conditions of the nervous system includes :

1. The Nervous and hysterical forms of palpitation.
2. Palpitation the result of some strong mental emotion.

*Nervous Palpitation.*—Nervous or hysterical palpitation is sufficiently common, and the condition of the system upon which it depends may be congenital, or acquired; it is more frequent in the female than the male, and in young persons than in advanced life; in the female too, it is often associated with deranged menstruation, leucorrhœa, spinal irritation, or hysteria. The palpitation is paroxysmal, sudden in its access, and liable to be excited by trifling causes; there are usually perfect intermissions, but the frequency of the attacks and their duration differ much in different subjects. The palpitation frequently comes on at night, particularly during the early part of the night, and prevents sleep; the patient's attention is constantly directed to his heart, and the attacks are accompanied by various disagreeable or painful sensations; sometimes there is a diffused pain in the left mammary region, sometimes this pain is limited to a space which could be covered by the palm of the hand, in other subjects the uneasy sensations are referred to the left axilla, and extend down the arm to the elbow on that side. The heart's action is habitually quicker than natural. The palpitation usually supervenes during repose, and is not felt when taking exercise, or when the mind is occupied: during its continuance, the sounds of the heart are increased in intensity, and are audible over a wider area than natural. In

the intervals the impulse is not increased, it may even be more feeble than natural.

The sensation of palpitation is sometimes referred to the epigastrium, sometimes to the region of the left scapula. In many instances the feeling experienced in the paroxysm, particularly if the patient is a female, is a disagreeable sense of fluttering in the region of the heart, or in the epigastrium, which, in poetic language, has been compared to the fluttering of a bird :

My fluttering heart  
Beats like a prison'd bird against its cage,  
When some annoying hand is stretched to seize it.

At others, it is rather a tumultuous action of the organ, with a sense of sinking, anxiety, or of oppression, attended by constriction at the epigastrium, or a sensation of choking, or shortness of breath. The digestion is frequently deranged, flatulence, borborygmi, and globus hystericus are common, accompanied occasionally by pain in the left side of the chest, increased by pressure or a full inspiration.

This condition of the system is often induced by close confinement, or excessive study, a too sedentary life, or prolonged mental exertions, by late hours, intemperance, and dissipation of all kinds, by the habitual use of strong tea, or excessive indulgence in smoking, by sexual excesses, or masturbation. The subject of nervous palpitation is often under the impression that he labours under organic disease of the heart; indeed it is a common observation that when the patient's attention is particularly directed to his heart, and when his complaint is principally of palpitation, the condition is more often one of functional derangement than of organic disease of the organ,

*Strong mental emotion* has a powerful influence upon the heart's movements, and has been recognised in all ages as a frequent cause of palpitation; indeed, from this circumstance, the heart was supposed by the writers of antiquity to be the seat of the passions. The heart is, however, differently affected by different kinds of mental emotion. The immediate effects of some as anger, joy, &c., are exciting, the heart's action becomes stronger, and the whole circulation feels its influence. The immediate effects of others as terror are, on the other hand, depressing, the palpitation is more of the passive form, and is more distressing to

the subject of it. Protracted mental emotion, as grief, anxiety, &c., as certainly but more slowly bring about analogous results.

The influence of mental emotion in giving rise to functional derangement of the heart is more marked in some constitutions than in others, and still more in some morbid conditions of the system than in health. Thus, in the anæmic state, in nervous, hysterical, and hypochondriacal subjects, in individuals debilitated by previous illness or other causes, strong mental emotion is more likely to be followed by derangement of the heart's action than when the individual had been previously in good health. Nevertheless, we not unfrequently meet with cases where a sudden shock, or fright, in a subject apparently in good health, is followed by permanent irritability of the heart; or where prolonged grief or anxiety more slowly bring about the same result. Indeed, Dr. Hamilton has made the remark, that palpitation of a very troublesome kind may arise from the impression made on the system by mere imagination.

Functional derangement of the heart symptomatic of other diseased states is observed:

1. In derangement of the digestive organs.
2. In the gouty diathesis.
3. In debility of the system.

*Dyspeptic palpitation.*—We have seen that organic disease of the heart is frequently associated with deranged digestion, of which it constitutes a very troublesome complication; on the other hand, dyspepsia itself, when protracted or severe, is a common cause of functional derangement of the heart. “Symptomatic affections of the heart are often (Dr. Abercrombie observes)\* among the most troublesome symptoms that accompany affections of the stomach, and always the most alarming to the patient.” Abernethy† likewise says that he had met many cases “where the great degree of palpitation led to a belief that some organic affection existed, which ceased on amendment of the general health; apparently arising from an amelioration of the state of the digestive organs, and the patient’s have continued in perfect health.”

In palpitation from this cause, flatulent distension, acid, or foetid eructations, headache, vertigo, or noises in the head are

\* On Diseases of the Stomach.

† On the Constitutional Origin of Local Disease.

common; and the patient frequently complains of a sensation of sinking at the præcordia, or of weight, oppression, or emptiness here. Epigastric pulsation is common, and there is often pain, or soreness in the præcordial region, experienced more when at rest than when in motion, which is rather relieved by pressure, and may or may not extend from the præcordial region to the left axilla or arm. The urine as it cools deposits a sediment, and not infrequently contains crystals of oxalate of lime.

The heart's action is readily excited, and is sometimes tumultuous in the paroxysm, it is not however increased, but is rather relieved by exercise, and no abnormal sound is audible on auscultation. The first sound, according to Dr. Corrigan,\* is often clearer and more resonant, though not louder than natural; he thinks that "this character is given to it by the neighbourhood of the larger cardiac portion of the stomach, which contains, as percussion indicates, a considerable quantity of air." The pulse is usually regular, though intermission is not uncommon.

The attacks of palpitation often supervene after meals, particularly breakfast, or at night after the individual has retired to rest; and this sometimes appears to have almost a mechanical origin: thus, when the stomach or intestines are distended by flatus, or undigested food, the descent of the diaphragm being impeded, the free expansion of the lungs and the movements of the heart are interfered with; hence arises palpitation and dyspnoea, particularly in the recumbent posture; or a distended colon might come to press upon the abdominal aorta, which, reacting on the left ventricle, would give rise to increased action of the heart in order to overcome it. Or, a distended stomach might press on the semi-lunar ganglion or solar plexus, or stretch or compress the nervous branches derived from the sympathetic, and so react upon the central organ of the circulation.

The irritation caused by the presence of worms in the alimentary canal, particularly the tœnia, occasionally gives rise to a very troublesome form of palpitation, which probably may be included under the head of dyspeptic palpitation.

*Gouty palpitation.* — The disordered action of the heart, which occurs in gouty subjects, is characterized by many strange and anomalous symptoms, exhibiting sometimes the features of

\* Dub. Jour. March, 1841.



organic disease of the heart, or simulating disease of other organs. It is most common in the *atonic*, latent, or irregular forms of the disease; it occurs also in patients who have suffered frequent attacks of the regular form, and whose health is broken down from this or other causes.

We have seen that gouty inflammation of the endocardium is an occasional cause of morbid deposit upon the valves, and about the orifices of the left side of the heart; and in some instances gouty palpitation would appear to have its origin in this chronic inflammatory condition of the membrane, when it more properly comes under the head of organic than of inorganic affections. Gouty inflammation is not, however, limited to the endocardium, the *pericardium* may also be its seat and be accompanied equally by palpitation; and it is not improbable, that the *white patches* so common upon the pericardial surface of the heart are, in some instances, due to gouty inflammation of the membrane. *Metastasis* of gout from the joints to the heart will also give rise to disordered action of the heart, but it constitutes a much more serious matter than the form we are considering, and is accompanied by far more urgent symptoms.

Gouty palpitation is most common in individuals in whom gout is hereditary, but who have never had a regular fit; or it occurs in the intervals between fits of gout, or it may be premonitory of a fit, and be relieved on the appearance of gout in the extremities. Gouty palpitation is usually preceded by or associated with derangement of the digestive organs, and it may be combined with general debility, and an enfeebled constitution, or with a plethoric habit. It is more common in males than females, though it is not unfrequent in females at the period of life when the menstrual discharge is about to cease.

The paroxysms of gouty palpitation present several varieties. The more sthenic form is characterized by irregular, or tumultuous action of the heart, with a painful sensation of weight, or constriction towards the centre of the sternum, or in the præcordial region, and a feeling of impending dissolution; the surface is pale and the pulse intermittent, or irregular. It commonly supervenes at night and during sleep, from which the patient awakens in a state of great alarm.

A paroxysm of the asthenic form is characterized sometimes



by a disagreeably painful sensation, as if the heart's movements were suddenly suspended, and the blood ceased for a moment to be transmitted by it, and immediately afterwards as if this fluid was propelled with augmented force to the brain; or by fluttering action of the heart, with intermission or irregularity of the pulse, faintness and pallor, followed by lassitude and depression. The attack may be induced by mental emotion or by physical exertion of some kind, such as hurried walking, stooping, or by indiscretion in diet.

In other instances, pain is rather complained of than palpitation, which may be trifling or so severe as to resemble angina; may be limited to the præcordial region, and to a space which could be covered by the palm of the hand, or it may radiate from this point to the left scapula and shoulder, sometimes to the right, or shoot through the chest, causing great distress and considerable alarm to the patient, who is impressed with the idea that he will die in one of the attacks. In other cases, the paroxysm resembles none of dyspeptic palpitation, there is usually, however, a greater amount of restlessness and irritability, and more frequently irregularity of the pulse. Gastric or biliary derangement with acidity, flatulence, and colicky pains, are common to all; and the immediate exciting cause of the paroxysm is often an indiscretion in diet.

Although gouty palpitation may be a purely inorganic affection, it often is not so; and when the symptoms enumerated above are marked and persistent, we not unfrequently have reason to suspect the existence of some dilatation of the ventricular chambers, or of some flabbiness of their parietes; or that excess of fat upon the heart or fatty (atheromatous) or ossific deposit in the arch of the aorta, or about the aortic valves, exists.

*Palpitation from debility.*—Debility of the system however induced, whether occurring in convalescence from acute disease, or the result of profuse discharges, is accompanied by increased irritability of the system, trifling mental or corporeal agents acting upon it more powerfully than in health, the heart's action is quickened, palpitation is readily excited and it presents many of the phenomena of anæmic palpitation. When debility is the result of exhaustion from sexual excesses, it is much more likely to be accompanied by morbid excitement of the heart than when

it supervenes slowly and is due to defective nutrition or similar causes. The variety which is the result of masturbation presents many of the characters common to the nervous, anæmic, and dyspeptic forms; it has been well described by Krimer.\* The palpitation is protracted and distressing, the action of the heart is violent, tumultuous, and irregular, accompanied by dyspnoea and tenderness at the epigastrium, with pallor, debility, lowness of spirits, amounting sometimes to hypochondriasis, nausea, deranged digestion, and other symptoms upon which it is unnecessary to delay. A severe form of palpitation occasionally occurs during convalescence from certain acute diseases; the action of the heart during the paroxysm being exceedingly violent, appearing in extreme cases to agitate the whole chest and giving the impression that it must be the result of organic disease. It is generally, however, perfectly removed by change of air, travelling, and tonic medicines.

Disordered action of the heart the result of disproportion between the capacity of the thorax and the size of the heart is observed:

1. In deformity of the chest from lateral curvature of the spine.
2. In distortion of the chest from angular curvature, the result of previous disease of the dorsal vertebræ.
3. In cases where the chest is from birth narrower than natural, or where this has supervened during the growth of the body.
4. In cases where the heart is congenitally smaller than natural.

*Palpitation from deformity of the chest* is observed in young subjects, particularly females, in whom lateral curvature of the spine in an extreme degree is present; as well as in individuals of either sex, who, in early life had been the subject of caries of the dorsal vertebræ. In both cases, the thoracic cavity is narrowed and contracted, and its shape altered according to the nature and amount of the deformity, while the organs contained in it may suffer compression or displacement, and the perfect expansion of the lungs, or the freedom of the movements of the heart, may be more or less interfered with.

In deformity of the chest, whether from caries of the spine, or independent of it, the contents of the thoracic cavity often adapt themselves so perfectly to the altered form of the parietes, that

\* Hufeland's Journal, 1827.

palpitation is not as often complained of as one would suppose from the amount of distortion present; and, in cases of lateral curvature of the spine, when palpitation is a prominent symptom, it has its cause as often in constitutional debility, in anæmia, or in functional derangement of the uterus as in the deformity. In some cases of distortion of the chest from caries of the spine, the movements of the heart are however considerably interfered with, palpitation in a greater or less degree is constantly present, and the impulse is felt and seen beyond its normal limits. Enlargement of the heart eventually ensues in such cases, and if the individual becomes the subject of acute inflammation of any of the organs contained in the thorax, the symptoms will be more urgent, dyspnoea and distress will be greater, and the disease will be more likely to terminate fatally than under opposite circumstances.

*Palpitation from narrowness of the chest.*—It occasionally happens that the chest from birth is smaller and narrower than natural, the thoracic walls are flattened laterally and bulge out in front, giving the sternum, with the costal cartilages, a keel-shape, and the capacity of the chest is diminished in proportion. Another condition which has some analogy with this, but is brought about in another way, is more frequently accompanied by symptoms simulating heart-disease. The condition to which I allude is observed in young persons about puberty, generally males, who are tall and thin, who have grown fast and as it is said, “outgrown their strength,” but in whom the chest from some cause has not expanded in proportion to the growth of the body. They suffer from palpitation, the heart’s action is more rapid than in health, and its impulse is seen and felt to be stronger and more diffused than natural, a larger portion of the organ coming in contact with the parietes of the chest; and the patient or his friends are impressed with the idea that organic disease of the heart exists. When we come to examine the organ, however, its sounds are found to be normal, the apex beat is felt nearly in its usual situation, and there are no signs of organic disease.

Under the name “functional derangement of the heart in growing persons,” Dr. Corrigan\* has described a variety which comes under this head. It occurs most frequently in growing boys, in whom the chest is narrow, or drawn in. Palpitation is

\* Dub. Jour. of Med. vol. xix.

the most troublesome symptom, it is increased by exercise so as to prevent the boy from joining in play with others, sometimes even the slightest exercise brings it on, or it comes on at night. The impulse of the heart is increased, and felt over a larger space than natural; "the contraction of the ventricle is *quick*," and the sounds are loud, but no abnormal sound is audible. The pulse ranges from 120 to 130. "There are occasionally cramps in the legs at night, which point to spinal or muscular debility. In some cases the symptoms have arisen at school, without any appreciable cause; in other instances, the disease has begun in boys who have been taken from a country life, or from school, and sent into an office, which has suddenly changed the boy's habits to a sedentary life."

*Palpitation from congenital smallness of the heart.*—A disproportion of another kind between the capacity of the thorax and the size of the heart occasionally exists; this organ may be congenitally smaller than natural, too small in fact to perform its functions perfectly. Congenital smallness of the heart according to Otto\* is sometimes connected with other vices of formation of the organ; at others, with general weakness, and imperfect development of the system, or of parts of it. It occurs however independent of any other morbid change, but the cases which have been satisfactorily recorded are few in number. The heart in certain chronic diseases accompanied by much emaciation, or where it has been compressed by tumors of the mediastinum, or the supply of blood going to it has been much diminished owing to obstruction, or obliteration of a coronary artery, undergoes *atrophy*, but this is quite a distinct condition from the one we are considering.

Under the head, *cor parvum*, Morgagni, Lieutaud, and Kreysig refer to cases where the heart after death, from various diseases, was found to be smaller than natural; these are however rather examples of atrophy of the heart. In modern treatises on diseases of the heart, the subject is passed over in silence, and the only recently recorded cases are, one by Dr. G. Bird,† one by Dr. Theophilus Thompson,‡ and one by Dr. J. Johnson,§ which were related at a meeting of the Medical Society of London, a few years

\* Path. Anat. Trans. by South.

† The Lancet, vol. ii. 1844.

‡ The Lancet, vol. ii. 1844.

§ Ibid.

since; with one which I communicated to the Surgical Society\* of Ireland.

Two of these cases were males, and two females. The symptoms were palpitation, which was present in three; fainting fits in two: in my case the patient had frequent fits of unconsciousness, in which the face and limbs became livid; the extremities were also habitually cold, and spots of purpura frequently appeared upon the lower extremities. In none of the cases was any abnormal sound audible in the region of the heart. The death was sudden in three, in one it occurred in a fainting fit, and in another, on making a slight exertion.

In neither of the three first cases are the weight of the heart, or the measurements of its several parts given. In Dr. Johnson's case, an adult male, the heart is stated to have been no larger than a pigeon's egg, and in Dr. Thompson's, to be peculiarly thin and small. In Dr. Bird's case, the patient was a female, aged twenty-four; the heart was not larger than that of a child of eleven years of age and its parietes were remarkably thin. In my case, the patient was a boy, nine years of age; the heart weighed only two ounces; it measured from the base of the auricles to the apex, two inches ten lines, and across the base of the ventricles two and a half inches. The cavity of the left ventricle measured thirteen lines in its long diameter, and nine lines transversely; the cavity of the right ventricle measured fourteen lines in its long diameter, and six lines transversely.

A form of "functional disorder of the heart," has been described by Dr. Christison,† which occurs chiefly during adolescence and in young adults, generally males, which is characterized by "constant violent pulsation in the præcordial region and præternatural heaving impulse;" the apex of the heart beats "between the fourth and fifth ribs very near to the sternum, and the dulness on percussion in the region of the heart is not so extensive and does not reach so low down as usual." The heart's sounds are diffused but no murmur is present. Several cases are related, and Dr. Christison seems to regard them as examples of congenital, or unusual smallness of the heart. To me they appear rather to belong to cardiac congestion, or nervous palpitation; if they are to be classed with inorganic affections of the heart.

\* Dub. Med. Press, vol. xxxvii.

† Ed. Month. Jour. February, 1845.

## TREATMENT.

Functional derangement of the heart, we have seen, may be brought about in a great variety of ways, and may be connected with very opposite conditions of the system; the treatment consequently must vary considerably in different cases. In the majority, it is a secondary affection, and our remedial measures must be directed to the removal of the primary affection, while in many instances, treatment is rendered difficult by the association of two or more morbid conditions, each of which by itself is sufficient to cause functional derangement of the heart. Our remarks under this head must, therefore, be somewhat general, we can do little more here than lay down the leading principles upon which the treatment is to be conducted.

*In the anæmic form*, the indications of treatment are to endeavour to diminish the watery state of the blood, and to increase the red corpuscles; to remove debility which is so constantly present; to tranquillize the nervous system, and calm inordinate action of the heart; and to remedy derangement of the uterine function, whatever be its nature. These objects will be attained, or at least will be greatly assisted by wholesome and nutritious diet, by tonics, particularly the preparations of iron, by exercise in the open air, by travelling, change of air, early hours, the tepid shower-bath, and the employment of medicines calculated to restore and improve the secretions. As the general health improves, and as the blood regains the healthy standard, the palpitation subsides, and the abnormal sounds in the veins and arteries diminish, and finally disappear.

The preparations of iron are far more efficacious than any other tonics in these cases, because in addition to their tonic action, they exert a direct effect upon the blood, increasing the red corpuscles, and supplying hæmatin, in which the latter are deficient. Any of the preparations of iron may be employed with this object, and every practitioner almost has his favourite formula. Some prefer the sulphate, either in pill combined with aloes or rhubarb, or in solution in water with the addition of a little dilute sulphuric acid. The muriated tincture in small doses, largely diluted, is however a better preparation, and the sulphate of quinine may, if considered advisable, be combined with it, without diminishing

the transparency of the mixture. The objection that the acid is liable to discolour the teeth, may be obviated by gargling the mouth, after each dose, with a weak solution of carbonate of soda—an agreeable mode of administering the muriated tincture is in effervescence with bicarbonate of soda, or potash, as originally employed by the late Mr. Carmichael. The iodide of iron is a favourite with many, and the syrup is an agreeable form. The saccharine carbonate, when well prepared, constitutes one of the best modes of exhibiting a protosalt of iron. The compound mixture, owing to the other substances which enter into its composition, is less agreeable, and the aromatic mixture contains too small a proportion of iron.

The combinations of iron with the vegetable acids, as in the ferro-citrate, ammonio-citrate, tartrate, and acetate, or in the form of double salts, as the potassio-tartrate, or citrate of quinine and iron, constitute convenient forms for the exhibition of iron. Indeed, the opinion has been advanced that the only preparations of iron useful in these affections are its combinations with the vegetable acids, or with carbonic acid; the salts which iron forms with the mineral acids being supposed to act rather as astringents. That this view is not correct, must be evident to all who have employed the latter extensively; certainly if given in large doses, their astringent and irritative properties would tend, as Dr. Turnbull\* observes, to prevent absorption; but, in small doses largely diluted, I prefer them to any other.

In order to counteract the stimulant property of iron, aloes is often combined with it, and the sulphate may be given in pill with aloes and an aromatic, or the aromatic mixture with the compound decoction of aloes; with the same object the vinum ferri may be given with the wine or tincture of rhubarb. In protracted cases, a visit to the chalybeate mineral springs in these countries, or on the continent, will contribute to the restoration of health.

The treatment of palpitation from *plethora* is directly the reverse of that for *anæmia*: here nutrition is active, blood is rapidly formed, and the vascular system is overloaded. The indications of treatment therefore are:

\* The Lancet, April, 1846.



1. To diminish the actual amount of blood, and to unload the vascular system by venesection, or local bleeding.

2. To prevent the too rapid formation of blood, by diminishing the daily allowance of animal food, restricting the individual to a spare diet, from which malt liquors and wine are to be excluded, and enforcing a due amount of exercise.

3. To relieve visceral congestion by the free use of saline or other purgatives.

4. When it depends upon the suppression of an habitual discharge, as from an issue, or old ulcer, to make an issue or seton in some other part; or if it depend upon the suppression of an hæmorrhoidal flux, to apply leeches occasionally in the vicinity of the anus.

When palpitation is the result of *local plethora*, or *cardiac congestion*, if the causes which have conduced to bring about this state can be entirely removed, the inordinate action of the heart will in most cases gradually subside; but, the longer they have been in operation, the longer in general will be the interval before the complete disappearance of this symptom. “When the functions of the heart have been disordered by violent causes acting on young persons in the prime of life, the irritability of the organ may continue (Dr. Calthrop Williams observes) for a length of time in spite of the most judicious treatment, and then suddenly disappear without our being able to account for the unexpected change. Attention to the state of the digestive organs is a point of great importance in the treatment of those who have suffered from congestion of the heart, and the practitioner should be careful to avoid having recourse to strengthening medicines as they are called, unless imperiously indicated; for these remedies often increase the morbid irritability of the heart instead of diminishing it.”

In *nervous* and *hysterical palpitation* with which general debility is often associated, our efforts are, in the first instance, to be directed to the removal of the cause; the indications then are to diminish constitutional irritability, and to correct the condition of the system upon which the functional derangement of the heart depends; to relieve flatulent distension and other dyspeptic symptoms when present; to correct morbid secretions, and to improve the general health. These indications will be best fulfilled by particular attention to diet, by early rising, and regular exercise



in the open air, by the use of the tepid or cold shower bath, or sea bathing, by change of air, travelling, cheerful occupation, agreeable society, and all such means as withdraw attention from the heart.

The purely medical part of the treatment, consists in the employment of antispasmodics, and diffusible stimulants for the relief of the paroxysm, as the aromatic or foetid spirit of ammonia, or the ammoniated, or simple tincture of valerian in camphor mixture, hydrocyanic acid, and the liquor opii sedativus, or solution of muriate of morphia, with ether, or Hoffmann, &c., according to circumstances, with aperients or laxatives when required, and tonics in the intervals. Among metallic tonics, Dr. Hope gave the preference to sulphate of zinc (as being the least stimulant) in grain doses, twice or three times a day, combined with extract of gentian; the valerianate of zinc is generally preferred now to the sulphate. In these cases however medicines will do little, unless preceded and accompanied by the other measures alluded to: "plain food, regular exercise, and early hours will do more (as Dr. J. Johnson quaintly remarks) than all the assafoetida, bark, and valerian in the apothecaries' shops."

*Gouty palpitation.* The treatment of gouty palpitation resolves itself into:

1st. The relief of the paroxysm.

2nd. The removal of the causes which have given rise to it.

In some instances, the functional derangement is premonitory of a fit of gout, and is relieved as soon as gout makes its appearance in the extremities. In other cases, temporary relief will generally be obtained from antispasmodics, carminatives, and anodynes, variously combined, or if offending matters are contained in the intestines, or flatulence of the colon is present, by enemata, containing assafoetida, turpentine, &c.

That excess of lithic acid is present in the system of gouty subjects, the result of mal-assimilation of the food, or of the continued use of certain articles of diet, or of its defective elimination by the kidneys and skin, admits I believe of no doubt. Dr. Garrod has detected its presence in the blood, and has, moreover, shown that it disappears from the *urine* immediately before, and during a regular fit, and re-appears as the latter declines. The preventive measures therefore resolve themselves into:

1st. To endeavour to remove the excess of lithic acid from the system.

2nd. To prevent fresh formation.

The first indication will be best fulfilled by the administration of alkalies, or alkaline carbonates, with colchicum, preceded when necessary by aperients, and followed by tonics. The alkaline carbonates are less likely to disagree than the pure alkalies, and potass is to be preferred to soda, owing, as Dr. Prout has shown, to lithic acid forming a soluble salt with potass, and an insoluble one with soda. Colchicum may be combined with the carbonate of potass, or given in the intervals. I have seen more benefit from the iodide of potassium in these cases, combined with the liquor potassæ, and tincture of the seeds of colchicum, than from the alkaline carbonates. The colchicum ought never to be administered in doses likely to occasion its depressing effects, the dose should rarely exceed five drops. The mineral saline waters, either in England or on the continent, when the patient can resort to the locality, are often of use in these cases, and furthermore, prove serviceable by the change of air and of occupation. If the patient's circumstances do not permit of his visiting these watering places, he may use the imported waters, or the imitations of them which are now manufactured on a large scale.

The second indication will be best fulfilled by strict attention to diet, by the avoidance of all articles of food which experience has shown to favour the generation of lithic acid, such as highly seasoned dishes, rich sauces, pastry, cheese, pickles, pork, salmon, &c., by the moderate use of animal food, by avoiding malt liquors and the stronger wines, by promoting the urinary and cutaneous secretions, by regular daily exercise, by the use of the tepid or warm bath, by frictions to the surface, and warm clothing.

In *dyspeptic palpitation*, when any tenderness on pressure exists at the epigastrium, leeches or counter-irritation will be required in the first instance; and if the bowels are sluggish, or offending matters are lodged in them, they must be unloaded. Dyspeptic palpitation is frequently associated with a gouty habit, in which acidity and flatulence are prominent symptoms, and relief to the latter is generally followed by relief of the former.

When gastric irritation has been relieved, or removed, bitters and tonics, with alkalies or alkaline carbonates, or with dilute

ids, according to circumstances, will be of use. Sometimes light bitters, as infusion of cascarilla, calumba, or orange-peel with an aromatic tincture, agree best; at others stronger bitters, gentian, quassia, or quinine, answer better. They may be given with kali, carrara, lime, or magnesia water. In acidity of the cœcum or colon, magnesia in substance forms the best antacid, the soluble alkalies (as Dr. Prout observes) being absorbed before they reach the lower bowel." If epigastric pain continues, the subnitrate of bismuth with soda and aromatic powder is often of service, and if irritability of the stomach exists, hydrocyanic acid constitutes the best sedative and antispasmodic, relieving pain, subduing gastric irritability, and calming the disordered action of the heart.

In the oxalic diathesis, Dr. Prout gives the preference to the mineral acids, particularly the muriatic and nitro-muriatic. "Their effects must however be watched, and when they begin to produce the deposition of lithate of ammonia, or of lithic acid, their use must be suspended." Strict attention to dietetic and hygienic rules constitutes a most essential item in the treatment; indeed the other measures alluded to will generally fail unless these are strictly carried out.

When *pain* referred to the left mammary region is much complained of, in this, or in other forms of functional derangement of the heart, a belladonna plaster worn for some time, often gives relief; sometimes counter-irritation is more effectual, and a blister about the size of a crown-piece may be applied to the painful point, and repeated according to circumstances. Dr. Corrigan prefers the croton oil liniment for this purpose, he believes there is something specific in its action, as he has not found the same beneficial effects from other counter-irritants. It must, however, be borne in mind, that "the functional derangement of the heart, and the uneasy symptoms referred to the præcordial region, may continue long after the cause has ceased to exist, or to be a prominent feature in the case." In many instances, months, Dr. Corrigan observes, have elapsed after the gastric or intestinal derangement which had been the original cause, have ceased to be troublesome; in some instances even years."

In that form of functional derangement of the heart which is observed in growing boys, when the expansion of the chest has

not kept pace with the growth of other parts of the body, ~~we~~ often find debility associated with it, particularly if antiphlogistic treatment had previously been used. It is in general relieved by change of air and change of occupation, by nourishing diet, tonics, particularly quinine and iron, sea-bathing, &c.

## CHAPTER XXIII.

ADVENTITIOUS DEPOSIT IN THE ARCH OF THE AORTA.—VARIETIES OF.—  
ALBUMINOUS AND SEMI-CARTILAGINOUS DEPOSIT.—ATHEROMATOUS  
(FATTY) AND OSSIFIC DEPOSIT.—EFFECTS OF, UPON THE AORTA, UPON  
THE HEART.—SYMPTOMS AND DIAGNOSIS OF.

THE aorta, particularly its arch, is very frequently the seat of morbid deposit, indeed we seldom examine this part of the artery in aged subjects without finding traces of it. It is not, however, limited to advanced life; it has been met with in infancy, and it is common in the middle periods of life, particularly in subjects prematurely old from intemperance. It is much more frequent in the male than the female.

Two principal forms of primary adventitious deposit are met with in the arch of the aorta:

1. The “albuminous” of Bizot; the “excessive deposition of the lining membrane of the vessels” of Rokitansky; which has its original seat *upon* the free surface of the lining membrane, and passes into the semi-cartilaginous deposit.

2. The fatty (atheroma), which is previously deposited *under* the lining membrane, and becomes the nidus for ossific deposit.

Both are met with in the same subjects; they are, however, independent of one another, and the ulterior changes in each are different.

These deposits are very generally attributed to *arteritis*, and are supposed to be the result of chronic inflammation of the inner coats of the artery. Writers, with few exceptions, have adopted this view, though there cannot be a more erroneous one—these deposits being altogether independent of inflammation. Indeed, if it were correct, chronic aortitis would be one of the most common affections, and few individuals would attain old age without having been the subject of it; whereas idiopathic inflammation of the lining membrane of the aorta is one of the rarest diseases

known. These deposits are simply forms of degeneration; the most common, and the most important in its ultimate results (atheroma), belonging to the class of fatty deposits, and constituting a variety of *fatty degeneration*.

These deposits are the source of almost all the morbid conditions of the aorta; dilatation of this vessel is an ordinary consequence, and aneurism is one of the secondary effects of the changes which they bring about; while rupture of the aorta never occurs without having been preceded by them.

#### ALBUMINOUS DEPOSIT.

The seat of this deposit, and the nature of the changes which it undergoes, were first correctly laid down by M. Bizot.\* According to him, this deposit on its first appearance, is a smooth, gelatinous-looking exudation upon the free surface of the lining membrane of the artery, generally first seen upon that part of the arch of the aorta where branches are given off. It occurs in patches of variable size and thickness, it is transparent or slightly coloured, but without any injection, or increase of vascularity in the parts about or under it. The patches can at first be separated from the lining membrane of the artery; after a time the union becomes so intimate, that the lining membrane comes away with them, the patches at the same time becoming opaque, and thicker from the addition of new layers.

In the advanced stage, these patches, in consistence and color, resemble boiled white of egg, or they are more dense and tough, and have nearly the consistence of cartilage. The lining membrane of the artery at their site disappears, and they are then in contact with the middle coat of the artery. They do not become the nidus for osseous or calcareous deposit, as supposed by some, but it occasionally happens that atheroma is deposited under the lining membrane at their seat, where it undergoes the changes presently to be described. The thickness and extent of this deposit differs in different subjects. "It varies in thickness from a quarter of a line to two lines and upwards, and extends in extreme cases over the whole trunk and main branches of the aorta, implicating the entire arterial system. The deposition is generally thickest directly over the division of a trunk, or at the bifurcation

\* Mem. de la Soc. Med. d'Observation, t. 1, 1836.

of a vessel. At these points the deposit is frequently so thick, that the mouths of the divergent vessels are much contracted, and even wholly closed.\* It is sometimes more abundant in the smaller arteries than in the aorta, and its effect necessarily is to diminish the elasticity of the vessel. Bizot supposed it to be the result of inflammation, but his view cannot be received now. This deposit when fully formed is generally described as cartilaginous, or semi-cartilaginous, but, according to Rokitansky, "it has nothing in common with cartilage, or fibro-cartilage; it consists of structures analogous to the layers which constitute the lining membrane of the vessel, viz., epithelium, fenestrated membrane, and longitudinal fibrous coat."

Rokitansky says that he has observed openings, or foramina, upon the free surface of this deposit which vary in size "from a pin's head to that of a poppy seed," and are numerous, or the contrary. They lead to canals which penetrate the deposit to various depths, and they "either terminate without changing their form or again divide, and enter the circular fibrous coat where they finally ramify."

#### ATHEROMATOUS DEPOSIT.

Atheroma makes its appearance as a minute, yellowish-white, more or less circular spot, not elevated at first, seated in the sub-erosus or sclerous coat of the artery, and consequently under the lining membrane to which it adheres, and which preserves its transparency at this period. If we detach the lining membrane the little patch comes away with it. The spots may be few in number, or the contrary, and they are often first seen where branches come off from the aorta. Atheroma once deposited does not remain stationary, the spots become more opaque, increase in size, or coalesce into larger and more irregular patches, forming light elevations upon the surface of the interior of the artery. The lining membrane is still perfect, but the patches have become more adherent to the middle coat.

The subsequent changes which this deposit undergoes may be included under the two heads laid down by M. Bizot, viz.:

1. Softening.
2. Ossification.

\*Rokitansky *Path. Anat.*, Trans. by Day, vol. iv.

**Softening.**—When softening commences, the opaque patches assume a yellow colour, enlarge, and become prominent; while the deposit becomes less consistent, being sometimes so liquid that it can be pressed from one side of the patch to the other; in general, however, it has about the consistence of putty, or purée.

The process of softening is not confined to the atheroma, the middle and inner coats of the artery both become involved. The layers of the middle coat in contact with the deposit become adherent to it, soften, and, according to Rokitansky, undergo fatty degeneration; the lining membrane being compressed by the increase of the deposit is eventually absorbed over the patches, or it cracks. The softened contents of some of the patches may become absorbed at this period, when the lining membrane will be depressed in the centre, or if they were numerous, the interior of the artery will present a wrinkled appearance. At other points, the lining membrane being destroyed, the softened atheromatous matter is washed away in the current of the blood, and the interior of the artery at these places has the appearance of being ulcerated. Although these are often termed ulcers, and were supposed to be the result of ulcerative absorption, they have no title to the name, as they are altogether independent of inflammation.

Gluge and Gulliver first pointed out the true nature of atheroma, and showed that it is a fatty deposit, under the microscope exhibiting oil globules and crystals of cholesterine. It constitutes, in fact, a form of fatty degeneration, and, as such, is not to be regarded simply as a local disease, but as part of a constitutional diathesis; we often, consequently, find it associated with fatty degeneration, or fatty deposit on the heart, or in other situations.

Softening of atheroma, involving the middle and inner coats of the artery, usually precedes the occurrence of aneurism, particularly in the lower extremities; and M. Bizot has shown that atheroma is often deposited *symmetrically*; thus, if present in one femoral or popliteal artery, it is likely to be found in the corresponding artery of the opposite limb. Hence, when a patient has laboured under aneurism in either of these vessels, the disease is liable to supervene on the opposite side, or in the aorta; as atheroma is seldom found in the arteries of the lower extremity without being likewise present in the aorta. Softening of atheroma



appears to be influenced in some respects by the age of the individual. "Once deposited, the younger the subject the greater would be (Dr. Blakiston\* says) the tendency to the formation of aneurism. For this depends not so much on the amount of atheroma as on its tendency to soften. In old age the reverse of this takes place; the atheromatous patches are dry, or are soon converted into calcareous concretions. By the same law, the softening of tubercular matter takes place with less and less rapidity as age advances; so that the older the patient the more rapid is the march of the disease."

*Ossific deposit.*—Atheroma frequently becomes the nidus for ossific deposit; and this would appear to be the mode in which nature seeks to repair the injury the artery has suffered, to preserve the integrity of the tube, and to strengthen the vessel at its weakest point.

The ossific deposit makes its appearance in the atheromatous patch as a minute dense point, which gradually increases in width, and ultimately forms a thin osseous plate or scale, of variable size, covered at first by the lining membrane; as it enlarges, the lining membrane of the artery is removed, and the osseous plate comes to be washed directly by the blood. At the same time, the layers of the middle coat in contact with it suffer from the compression; but this coat is seldom entirely destroyed, a thin layer, where it joins the cellular coat, as M. Bizot remarks, almost always remaining.

When atheroma is deposited under the semi-cartilaginous patches, it may either soften, or become the nidus for ossific deposit; and the ossific matter may then, by its increase, cause absorption of the semi-cartilaginous patches, and appear upon the interior of the artery, which has given rise to the supposition that osseous is always preceded by cartilaginous deposit." But osseous deposit has always atheroma for its nidus, and it is consequently always primarily deposited under the lining membrane; whereas the semi-cartilaginous deposit has its seat always upon the surface of the inner coat of the artery. The osseous plates when formed, are generally pretty firmly adherent; they sometimes however loosen, project upon the inner surface of the vessel,

\* On Diseases of the Chest.

and lead to rupture of the artery upon some sudden exertion on the patient's part. "Their number and size are subject to great variety; in some cases they only adhere at detached points; while in others the artery appears to be converted into a more or less solid osseous tube."

Morgagni compared these bony scales, where the interior of the artery is studded with them, to drops of white wax suddenly cooled. They resemble ordinary bone in hardness and density, as well as in composition, consisting of phosphate of lime and animal matter. They differ from true bone in wanting the lamellated structure, and in the absence of bone-corpuscles, and medullary canals, as shown by Valentine and others.

*Effects of adventitious deposit.*—These several deposits are most common, and usually most abundant, in the ascending portion of the arch of the aorta, and they may all exist simultaneously in different stages of progress in it. Their *primary* effects, when the interior of the vessel is thickly studded with them, are:

1st, To destroy the naturally smooth and polished surface of the lining membrane, and to render the interior of the artery uneven, rough, and irregular.

2nd, To diminish or destroy the elasticity of the coats of the artery, and to render the vessel rigid, so that its sides cannot readily be approximated.

When the lining membrane of the arch of the aorta has become rough and irregular, the passage of the blood over this rough surface causes certain modifications in the sounds, presently to be noticed.

When the elasticity of the coats of the artery has been destroyed, the vessel can no longer expand or contract with the varying amount of blood it receives at different periods of the heart's action; hence, when the ventricular systole ceases, the ascending portion of the arch would be partially unfilled if the blood from the large vessels which come off from its transverse portion did not regurgitate to occupy it. The recoil of the blood and its passage over the same rough surface gives rise likewise to certain modifications in the sounds, presently to be described.

The *secondary* effects of this morbid deposit are exerted partly upon the aorta and partly upon the heart:

Upon the aorta, by bringing about dilatation of the ascending portion of the arch and of the sinuses of Valsalva.

Upon the heart, by causing dilatation with hypertrophy of the left ventricle, and by leading to permanent patency of the aortic valves.

*Dilatation of the aorta.* Among the secondary effects of this morbid deposit, dilatation of the aorta is frequent; and the ascending portion of the arch and the sinuses of Valsalva are the parts in which it is generally most marked. Dilatation of this artery has been hitherto always attributed either to the increased force with which the blood is propelled into it by an hypertrophied left ventricle, or to some impediment to the circulation through the trunk of the artery, or in its terminal branches, where the elasticity of its coats has been impaired by previous disease. This theory, however it may account for dilatation at that part of the arch of the aorta which receives principally the shock of the blood transmitted by the left ventricle, is quite insufficient to account for dilatation of the commencement of the ascending portion of the arch, or of the sinuses of Valsalva. The dilatation here is the result of the distension, caused by the regurgitation of the blood at each ventricular diastole, from the non-elastic portion of the arch above, and from the large arteries which arise from its convex surface; and the sinuses of Valsalva necessarily participate in the dilatation.

It has been long known, that in old age the lower part of the ascending portion of the arch of the aorta is generally somewhat dilated, it does not form a perfect cylinder, it advances further, and projects more to the right side than natural. This never could be brought about (as is generally laid down) by the continued force of the impulse of the blood transmitted by the left ventricle; its cause evidently lies in the regurgitation of the blood from the upper part of the arch into this the most depending portion, owing to the impairment of the elasticity of the coats of the vessel which occurs in advanced life. Indeed, even in health, there must be a slightly backward current of blood here, at each ventricular diastole, in order to close the semilunar valves.

Owing to the increased force required to transmit the blood through the rigid and unyielding aorta, the parietes of the left ventricle eventually become increased in thickness; and its chamber

becomes dilated. While the long continuance of this state may lead to retroversion of the free margin of one of the semilunar valves; these valves, at each ventricular diastole, being exposed to the shock of a regurgitating current of blood, yield, and eventually permit regurgitation, which adds considerably to the gravity of the case, and disposes to a fatal termination at a much earlier period.

#### SYMPTOMS.

This morbid condition, when the disease is fully developed, is characterized by certain well-marked signs, which enable it to be diagnosed with facility; yet in works devoted especially to cardiac pathology, either no attempt is made to trace its symptoms, or those which are set down to it are anything but pathognomonic. That its symptoms are positive and characteristic, and that they are such as directly proceed from the morbid state of the aorta, I shall endeavour to show; and I may add that these symptoms are not laid down here for the first time, they were embodied in an account of the disease communicated by the writer to the Surgical Society of Ireland, in March, 1847, and published in the *Dublin Medical Press* for April, of the same year; and subsequent experience has confirmed in every respect the correctness of the views then set forth.

These symptoms are :

1. Visible pulsation in the large arteries of the neck and upper extremities.
2. Jerking and receding pulse, resembling that of aortic regurgitant disease.
3. A short, double, sawing murmur, audible on auscultation over the first bone of the sternum, but inaudible at the apex of the heart.
4. An impulse perceptible to the ear laid on the stethoscope, over the first bone of the sternum, if dilatation of the aorta has supervened.

*Visible pulsation* in the large arteries is very marked in the carotids and subclavians; and in these vessels, it is sometimes a more prominent symptom than in regurgitant disease of the aortic valves. The radical pulse is usually likewise locomotive.

*The pulse* is jerking and receding but regular, presenting all

the characters of the pulse of unfilled arteries, and resembling accurately that of aortic regurgitant disease. These characters of the pulse become more evident if the finger is laid upon a larger artery than the radial, as the brachial, and if two or three fingers be placed upon the line of the artery. On making pressure upon the carotids at the root of the neck, a fremitus is usually likewise felt.

When the stethoscope is laid upon the first bone of the sternum, *short, double, sawing, or rasping murmur* is heard, which is audible from the upper edge of this bone to the junction of the costal cartilages of the fourth ribs with it, diminishes in intensity on either side of the sternum, and is never audible at the apex of the heart. The first of these morbid sounds is synchronous with the systole, the second with the diastole of the ventricles; both appear to be superficial, and near the ear, and both are short and rough, never prolonged or blowing. In the carotids at the root of the neck, a double sound is audible, which can be converted into a double murmur by making pressure with the stethoscope; it, however, has not the characters of the former, being harsh and somewhat blowing.

When the arch of the aorta, in addition to being rigid and inelastic, is *dilated*, an impulse, synchronous with the pulse, will be perceived when the ear is applied to the stethoscope laid upon the first bone of the sternum. This, unless the vessel is considerably dilated, is not sufficient to communicate an impulse to the hand laid upon the part, but is very perceptible to the ear laid upon the stethoscope.

The foregoing signs, when combined, are pathognomonic of this morbid condition; they will be better marked, or the contrary, according to the roughness of the interior of the artery, according to the force with which the blood is propelled by the left ventricle, and as this fluid is attenuated or the contrary.

#### MECHANISM OF THE PRODUCTION OF THE SYMPTOMS.

The mechanism by which these signs are produced admits of a ready explanation. The arteries in the normal state are lined, we know, by a smooth and polished membrane, over which the blood glides without producing any sound appreciable to the ear. At the same time these vessels are capable of adapting themselves

to the varying amount of blood they receive at different periods of the heart's action, and are always filled whether the quantity be greater or less. This depends upon a peculiar property possessed by arterial tissue, usually termed elasticity, but evidently something more than mere elasticity, which is common to dead as well as living tissues. The aorta in its healthy state possesses this vital property in a high degree, which is due not only to the proper fibrous coat, but to its subserous or *sclerous* coat, which in this vessel (as Dr. Chevers\* has shown) is particularly well developed.

In the morbid state we are considering, the lining membrane of the aorta being rough and uneven, the passage of a current of blood over this rough surface during the ventricular systole is attended with increased friction, and a murmur is developed which is audible of course at the period of the *first* sound of the heart; is loudest immediately over the part of the aorta in which it is developed, that is, at the first bone of the sternum; and is rough and harsh in proportion to the unevenness of the lining membrane, and the strength of the systolic current.

When the ventricular systole ceases, the artery, owing to the rigid inelastic state of its coats, is incapable of contracting upon its contents, and a partial vacuum would be created here if the blood from the large arteries which arise from the transverse portion of the arch did not regurgitate to occupy it. The passage backwards of a current of blood over this rough surface during the ventricular diastole develops a murmur which is heard of course at the period of the *second* sound of the heart, and is rough and harsh according to the roughness of the lining of the artery and the strength of the regurgitating current.

When the interior of the arch of the aorta has not lost its natural smoothness, although its coats are rigid or inelastic, a double sound is heard at the same part of the thorax, but unaccompanied by any murmur; it rather resembles the double sound of the heart transmitted to the ear at this point, for which it is commonly taken. This is often observed in old age, at which period we know the elasticity of the coats of the arteries is generally impaired, and the ascending portion of the arch of the aorta becomes usually somewhat dilated.

\* Guy's Hosp. Rep. No. 10.

The jerking pulse, and the visible pulsation in the large arteries of the neck, are due (equally as where the aortic valves permit regurgitation) to the unfilled state of the vessels. Here, however, regurgitation occurs into the aorta itself, not into the left ventricle. These signs are better marked when the ascending portion of the arch of the aorta, in addition to being rigid and inelastic, is at the same time somewhat dilated, the vacuum being more considerable, and a larger amount of blood necessarily regurgitating.

*Diagnosis.*—The diseased condition of the arch of the aorta under consideration is by no means uncommon, and has been long familiar to pathologists. Its symptoms, too, as we have seen, are sufficiently characteristic, yet they were entirely overlooked until laid down by the writer a few years ago; indeed, no writer upon diseases of the heart seems to have been cognizant of the fact, that regurgitation into the ascending portion of the arch of the aorta from the large vessels arising from the convexity of the arch, occurs under any circumstances. This has arisen partly from the symptoms in the early stage not being such as to attract particular attention, and partly to the circumstance that this morbid condition of the aorta is ultimately succeeded in many instances by regurgitant disease of the semilunar valves; and the latter lesion being found after death, the symptoms have been referred exclusively to it.

The valvular lesion, for which this morbid condition of the aorta is commonly mistaken, and with which it has hitherto been confounded, is disease of the aortic valves permitting regurgitation; or a combination of regurgitant with obstructive disease of those valves, which it resembles in the visible pulsation of the large arteries of the neck and upper extremities, in the peculiar character of the pulse, and in a murmur being audible in each on auscultation. They are distinguished by the tone and other characters of the murmur, by the situation in which each is best marked, and by the manner in which each is transmitted to the ear. Thus, in regurgitant disease at the aortic orifice, the murmur is single, prolonged, soft, and blowing; it accompanies the ventricular diastole only, and is loudest at the apex of the heart. When obstruction is combined with regurgitation, the murmur with the first sound is prolonged and blowing, or whistling,

occasionally rough and harsh ; it is, however, loudest at the base of the heart, from which it is propagated in the course of the current from the ventricle. In the morbid condition of the arch of the aorta we are considering, the murmur is always double, short, and sawing, or rasping, *never* single, prolonged, or blowing; it is loudest over the first bone of the sternum, is not propagated in the course of the current of blood from the ventricle, and is never audible at the apex of the heart.

The diagnosis between these morbid states is one of considerable practical importance, because the diseased condition of the aorta, which has been described, is not one which perils life in the same degree as disease of the semilunar valves permitting regurgitation: a patient labouring under it may, with ordinary care, live for many years, and eventually die of some other affection; whereas the subject of aortic regurgitant disease is never long lived; the death too is frequently sudden, and directly or indirectly the result of the valvular lesion.



## CHAPTER XXIV.

**THORACIC ANEURISM.—VARIETIES OF.—TRUE ANEURISM.—FALSE ANEURISM.  
MODE IN WHICH THORACIC ANEURISM FORMS.—PROGRESS AND TERMINA-  
TION OF.—SPONTANEOUS CURE OF.—SITUATIONS IN WHICH THORACIC  
ANEURISM MAY RUPTURE.**

**THE** term Aneurism, from *ανεκφυρω*, to dilate much, was originally applied to every increase in the calibre of an artery. The term is now, however, usually restricted to cases in which a partial and lateral dilatation of an artery occurs; while under *dilatation* are included the cases in which the entire circumference of the artery is dilated. As these terms have, however, been somewhat differently applied, it will be necessary to premise a short description of the several forms which occur in the thoracic aorta, and which have been distinguished by writers.

The most generally received division of spontaneous aneurism (in contradistinction to traumatic, which does not occur in the aorta) is into *true* and *false*, and is founded upon the supposed pathological condition of the coats of the artery at the part.

By the term *true aneurism*, is commonly understood that form in which all the coats of the artery enter into the formation of the aneurismal sac. By the term *false aneurism*, that in which the aneurismal sac is formed by the external, or cellular coat of the artery, after rupture, or destruction of the internal and middle. True aneurism is further subdivided, as the entire circumference of the artery is equally dilated, or as the dilatation exists at one side, in a *saccular* form.

The following table includes the several varieties, met with in the aorta, which have received distinct names:

True Aneurism	{	Cylindrical.	False Aneurism	{	Sacculated.
		Fusiform.			Mixed.
		Cirroid, or			Dissecting.
		Arterial Varix.			Varicose.
		Sacciform.			Hernial.
		Secondary Sacciform, or Multilocular.			

## TRUE ANEURISMS.

1. The *cylindrical* is that form in which the entire circumference of the artery is involved in the dilatation, and the limit between the dilated and undilated portion of the tube is abrupt.

2. The *fusiform* is that in which, while the entire circumference of the artery is dilated, the transition from the dilated to the undilated portion of the tube is gradual.

3. The *cirsoid* of Cloquet, the arterial varix of Dupeyren, is that form in which a considerable portion of the tube of the artery is engaged in the dilatation; the tortuous and dilated artery resembling in this respect a varicose vein.

The cylindrical and fusiform varieties are rather forms of *aneurismal dilatation* than of aneurism. The *cirsoid* aneurism is scarcely ever seen in the thoracic aorta.

4. The *sacciform* aneurism is that in which, in addition to circumscribed dilatation of the entire circumference of the artery, a lateral bulging takes place at one point, which is considered to be formed by all the coats of the artery, but in which, as it enlarges, they can no longer be traced.

5. The *secondary sacciform* aneurism, the multilocular aneurism, “l'aneurism sous l'aspect d'ampoules à bosselures,” of Cruveilhier, is that form in which several partial sacciform and secondary pouches form upon a cylindrical, fusiform, or sacciform aneurism.

## FALSE ANEURISMS.

1. The *saccular* aneurism is that form in which the dilatation is lateral and sacciform, formed by the external coat of the artery, after softening and absorption or rupture of the internal and middle coats, without any necessary dilatation of the artery from which it springs.

2. The *mixed* aneurism is that form in which rupture of the internal and middle coats supervenes upon true aneurism, the sac springing from some part of the circumference of the dilated vessel, and being formed by the external coat of the artery alone. Occasionally more than one sac springs from a cylindrical or fusiform dilatation, when it resembles the multilocular or secondary sacciform aneurism.

3. The *dissecting* aneurism, first noticed by Morgagni, and

described by Laennec, is that form in which, after rupture of the internal and middle coats, the blood, instead of distending the external coat into a sac, passes down between it and the middle coat, separating them around a portion of the circumference of the tube to a certain distance. In a few instances, the blood has passed between the inner and middle coats; or, after passing a certain distance between the coats, it has re-opened again into the artery lower down. The latter form was first noticed by the late Mr. Shekleton. The dissecting aneurism appears to be only a form of partial rupture of the aorta, the result of previous disease of the inner and middle coats.

4. The *spontaneous varicose* aneurism, is that form in which the sac of an aortal aneurism contracts adhesion with the superior vena cava, and finally opens into it. It is, however, rather a mode of termination of aneurism of the aorta by rupture in a particular way than a distinct form of aneurism; and it may be classed with the cases in which an aortal aneurism contracts adhesion with the pulmonary artery, or with the auricles of the heart, and opens into them.

5. The *hermial* aneurism is that form in which rupture of the middle coat of the artery alone takes place, and the inner coat is protruded through the aperture in the middle, and lines the sac formed by the external coat. It is a rare form of aneurism. The sac is necessarily small, as when it increases in size rupture of the inner coat must take place.

#### MODE OF FORMATION OF ANEURISM.

Aneurism never occurs in a perfectly healthy aorta; it is always preceded by adventitious deposit of some kind; and upon the changes which ensue as the result of this deposit, the several forms depend. Although aneurism is always preceded by adventitious deposit, it is comparatively a rare result of it; and it is even less likely to supervene when the deposit is thickly and widely diffused than when the patches are few and scattered. "Arteries which are thickly beset with old and unyielding deposits, and are thereby rendered inelastic, rigid, and brittle, are physically not in a condition (Dr. Chevers\* observes) to become the seat of diverticular aneurisms, formed by the yielding of their tissues."

\* Lond. Med. Gaz. 1845.

There appear to be three modes in which aneurism may form in the aorta, as the result of adventitious deposit; and they may be all included under the heads, true, false, and mixed aneurism.

1. *True aneurism*.—When the coats of the aorta are thickened, rigid, and inelastic from adventitious deposit in great part of its circumference, while they are still pliable and yielding at one point, the impetus of the blood against this point may distend it into a pouch; the middle coat of the artery, owing to the continued pressure, becomes thinned at this place, the pouch increases in size, and constitutes an aneurismal sac. This is the genuine *true* aneurism, and the manner in which it is developed was first correctly laid down by Dr. Chevers.\*

This form of aneurism is most common in the ascending portion of the arch of the aorta, or where the ascending joins the transverse; and it is always preceded by dilatation of the aorta at the part. The aperture by which the sac communicates with the artery is large, it may be of the same diameter as the sac, and the latter never acquires the flask shape of the false aneurism.

2. *False aneurism*.—When softening of atheroma, involving the middle and inner coats, has ended in destruction of these coats at one point, the distensile force of the blood acting upon the external cellular coat causes protrusion outwards of it, in the form of a sac. This constitutes the genuine *false* aneurism. It is observed in the arteries of the extremities more frequently than in the aorta; and the immediate cause of the giving way of the inner coats is not unfrequently a sudden muscular exertion, or an injury of some kind.

This form of aneurism is met with at every part of the aorta, its progress is commonly slow, and it seldom acquires a very large size. The sac has more or less of a flask shape, and we distinguished in it a body and a neck. The artery at the point from which the aneurism springs is not necessarily dilated, and its interior may present but few patches of atheroma. The orifice by which the sac communicates with the artery has more or less of a circular shape, and its edges eventually become smooth and rounded, giving it the appearance almost of a natural aperture.

\* Lond. Med. Gaz. 1845.

The interior of the sac in advanced cases is always studded with adventitious deposit, and is lined by a thin adventitious membrane. first described by M. Bizot, which extends into it from the artery, and appears at first sight to be the lining membrane of the artery continued into the sac.

3. *Mixed aneurism*.—This form commences as aneurismal dilatation; the middle and inner coats of the artery eventually give way at a point where atheroma has undergone softening, or where they have been thinned by the pressure and a secondary pouch is formed; or this may occur at several points in succession, when an irregularly dilated sac is formed. Under this head, the multilocular, and the secondary sacciform aneurism of authors may be included.

This is a common form in the aorta, but is rare in the arteries of the extremities. The interior of the sac is always rough and irregular from adventitious deposit, every variety of which is generally present in it; and it may, equally as false aneurism, be lined by a thin adventitious membrane. Its parietes are irregularly thickened in some places, and thinned in others. The body of the sac seldom contains much fibrin, but the secondary pouches are often lined or partially filled by it. This form of aneurism frequently acquires a large size; and, when several diverticula form upon it, distinct organs in the cavity of the chest may be compressed at the same time.

#### PROGRESS AND TERMINATION OF THORACIC ANEURISM.

When aneurism has once formed it seldom remains stationary; it generally has a constant tendency to increase in size, and this increase is usually at that part of the sac where the resistance is least; the distending force from within continually acting, the sac becomes gradually more and more distended, its parietes thinned, and it gives way at the weakest point. The progress of thoracic aneurism is, however, variable, being sometimes rapid, sometimes slow; and the rapidity or slowness of its progress is influenced by a variety of circumstances; more particularly:

1. The part of the artery from which aneurismal sac springs.
2. The nature of the parts with which it had contracted adhesion.
3. The form of the aneurism, its size, and immediate cause.

4. The thickness or thinness of the parietes of the aneurismal sac, and the presence or absence of fibrin in its interior.

5. The age and occupation of the patient, and whether his habits are intemperate or otherwise.

6. Whether the aneurism is confined within the walls of the chest, or forms a tumor externally.

7. Whether the parietes of the left ventricle are hypertrophied and dilated or the contrary.

As the aneurismal sac enlarges, the parts in its proximity which are encroached upon, are variously affected according to their nature and the amount of compression they suffer, lymph is generally effused upon the exterior of the sac, and adhesion takes place between it and the surrounding parts. The sac, as a result, becomes thickened; and this appears to be one of the modes by which nature seeks to strengthen the sac and to retard its rupture.

When the sac comes to stretch or compress *serous membrane*, the irritation excited gives rise to exudation of lymph, ending in adhesion between the sac and this membrane. Thoracic aneurism in this way frequently contracts adhesion with the pleura, and through it with the lungs. When the sac enlarges much, interstitial absorption takes place at the most prominent point, until the sac and the serous membrane, being thinned and stretched, crack at this point, and rupture occurs into the cavity of the pleura, or through it into the pulmonary tissue.

When the aneurismal sac comes to press upon a *hollow organ*, as the trachea, œsophagus, or one of the large bronchi, lymph is effused, and the sac contracts adhesion with it, followed by flattening and diminution in the calibre of the tube. The rings of the trachea in the course of time become absorbed at the point of pressure, the mucous membrane lining the tube undergoes a process of softening at the part, and the pressure continuing, a slough forms and rupture into the tube occurs.

When the aneurismal sac contracts adhesion with a large *artery*, as the subclavian or carotid, and compresses it, the calibre of the tube is, in the first instance, diminished; as the pressure increases, the irritation it excites gives rise to exudation of lymph in the interior of the artery, and the tube ultimately becomes impervious at the point, just as when a ligature is applied to a healthy vessel.

When the sac contracts adhesion with the superior *vena cava*, and compresses it, certain symptoms, presently to be mentioned, are observed; if the pressure increases, interstitial absorption in the intervening parts may go on until the aneurism opens into the vein. If the sac had contracted adhesions with the pulmonary artery or with the auricles or ventricles of the heart, the same result may follow.

When the aneurism, as it enlarges, comes in contact with *bone*, and contracts adhesion with it, the sac in the course of time is absorbed at the centre of the point of contact as well as the periosteum covering the bone; the latter is then washed directly by the blood, and forms a part of the parietes of the aneurism, and the continued action of the blood upon the bone ends in its erosion. In this way the bodies of the vertebræ, the sternum, and the ribs are partially or completely destroyed: the vertebral canal may even be opened, and rupture of the aneurism may take place into it. The inter-vertebral substance and the cartilages of the ribs, owing to their elasticity, resist the action of an aneurism much longer than bone.

When the aneurism reaches the surface and forms a tumor *externally*, interstitial absorption of the parts between the sac and the integuments gradually goes on; the pressure and distension increasing, the integuments at the most prominent point assume a dusky hue, which eventually passes into gangrene. As the slough becomes partially detached, hæmorrhage occurs, which may be temporarily arrested by the fibrin lining the sac acting as a kind of plug; but this is eventually detached, and fatal hæmorrhage ensues.

Aneurism of the aorta does not, however, often terminate by rupture externally; internal rupture is much more common. It may happen when the aneurism has formed a tumor externally, that the sac gives way under the integuments, and the blood is diffused in the cellular tissue upon the outside of the thorax. A case where this took place, was some time since under the writer's care.

When aneurism occurs in a subject in whom the left ventricle of the heart is hypertrophied and dilated, its progress is said to be more rapid than under opposite circumstances. Hypertrophy of the ventricles is not, however, a frequent complication of thoracic aneurism; fatty deposit upon the heart, as the writer pointed

out many years ago, is more common; and fatty degeneration of the tissue of the heart is occasionally present in addition, which is only what might be expected, seeing that the deposit which generally leads to aneurism, constitutes a form of fatty degeneration.

When thoracic aneurism occurs in a subject whose chest is broad and capacious, the tumor is more likely to increase quickly, and to attain a large size before attracting attention, than when it occurs in a subject whose chest is narrow and contracted, there being less resistance to the growth of the tumor in the former than the latter.

When an aneurism shows itself soon after a severe strain or injury of the chest, its progress is generally more rapid than when it arises independent of injury, though to this there are exceptions. It is also more likely to run a rapid course if the patient is intemperate, or if he is obliged to labour for his bread, and is unable to take the repose necessary to maintain the circulation in a tranquil state. Its progress, in this respect, is however capricious. I have known the disease to run a slower course when the patient was in the habit of drinking and of using physical exertions, than where all necessary precautions had been taken.

The progress of the disease is influenced, in some respects, by the part of the artery from which the sac springs. Thoracic aneurism is most frequent in the ascending and transverse portions of the arch, and it more frequently springs from the convex than the concave margin of this part of the artery.

When the aneurism arises from the portion of the aorta within the pericardium, it seldom attains a large size, rupture generally occurring into the pericardial sac while it is still small, and before it has made injurious pressure upon any important part. When the aneurism springs from the anterior aspect of the upper part of the ascending aorta, or of the transverse portion of the arch, it soon comes to act upon the sternum and ribs, and it points generally to the right of the sternum. When it arises from the convex margin of this part of the artery, it either causes erosion of the first bone of the sternum, or it rises up in the neck, or it points below either clavicle.

When it springs from the inner aspect of the ascending portion of the arch, it may contract adhesion with the pulmonary artery,



or with the left auricle, and may open into the pericardium, or into one of these parts. When it arises from the posterior surface of the transverse portion of the arch, or from its concave margin, it generally soon comes to compress the trachea, œsophagus, or bronchi; the vagus and recurrent nerves on the left side are very likely to suffer; and the superior cava, or right pulmonary artery are often compressed.

When the aneurismal sac arises from the *descending thoracic aorta*, none of the foregoing parts are likely to suffer compression; and the symptoms of aneurism of this part of the artery are as different from those of aneurism of the arch, as if they were distinct affections. An aneurism in this situation cannot form a tumor externally, until it has attained a very considerable size; it may, however, by pressing against the posterior surface of the heart, and protruding it forwards, give rise to palpitation, with increased action of the organ, and simulate hypertrophy of the ventricles; and it is almost certain to contract adhesion with the bodies of the dorsal vertebræ, and to cause erosion of the bone here, which is usually attended with very severe pain; and the latter may be almost the only symptom of its existence, until it forms a tumor externally.

#### SPONTANEOUS CURE OF ANEURISM.

While the foregoing changes are taking place upon the outside of the sac, another process is going on in its interior, by which rupture is delayed, the fatal hæmorrhage, when rupture has occurred, is retarded, and a spontaneous cure occasionally brought about.

This process consists in the gradual separation of the fibrin of the blood in its passage through the sac, and its deposition in concentric layers in the interior of this part; it is favoured by the roughness of the lining of the sac, by the aperture with which the sac and artery communicate being small, by the sac coming off from the artery at an acute or right angle, and if the latter has a flask shape with a narrow neck, and is of moderate size. Fibrin is not deposited in general dilatation of the aorta, and seldom when the orifice of communication between the artery and the sac is nearly of the same size as the latter, or when its interior is smooth.

Under favourable circumstances, the deposition of fibrin in concentric layers may go on until the sac is filled, the blood no longer finding an entrance, pulsation ceases, and a spontaneous

cure takes place. A case in which this occurred has been recorded by the writer.\* A spontaneous cure is however more rare in the aorta than in the arteries of the extremities, because there are several modes by which it may be brought about in the latter, while in the aorta there is but the one, viz., the deposition of fibrin in successive layers in the interior of the sac, until it is filled and no longer permits the entrance of blood. The outer layers of fibrin, or those next the parietes of the sac are, in old cases, hard, solid, and almost decolourized, while those nearer the centre have a red colour.

The deposition of fibrin seems to be favoured by the sac contracting adhesion with, and compressing the trachea. In two cases of the kind which were under the writer's care, in each of which death occurred without rupture of the sac, the aneurisms had almost undergone a spontaneous cure, the sacs being in great part filled with concentric laminæ of fibrin, of firm consistence and light colour, and evidently of long standing.

#### SITUATIONS IN WHICH THORACIC ANEURISM MAY RUPTURE.

From what has been said, we can understand how thoracic aneurism may rupture in many different situations. Thus it may open into:

1. The pericardium, the right or left auricle, or the right or left ventricle.
2. The pulmonary artery, or descending vena cava.
3. The pleura upon either side, or either lung.
4. The trachea, large bronchi, or œsophagus.
5. The posterior mediastinum.
6. The vertebral canal.
7. The cellular tissue upon the outside of the thorax, after having made its way through the ribs, or sternum.

Lastly, it may rupture upon some part of the surface; internal rupture is however much more frequent.

Rupture into the *pericardial sac* is common in aneurism of the ascending portion of the arch; indeed, Mr. Crisp's† valuable table of cases shows that this is the most frequent seat of rupture in aneurism of this part of the aorta. Thus, out of ninety cases tabulated by him, rupture into the pericardial sac occurred thirty

\* Dublin Med. Press, Dec. 17, 1845.

† On Diseases of the Blood Vessels.

times ; and out of forty observed by Dr. Swett,\* rupture in this situation occurred in seven. In aneurism of the transverse portion of the arch, rupture into the pericardium is much more rare ; thus, out of forty-eight cases in Mr. Crisp's table, it occurred only twice, and in thirty-one given by Dr. Swett, it occurred but three times.

Rupture into the *chambers of the heart*, is almost limited to aneurism of the ascending portion of the arch ; thus, in the ninety-eight cases tabulated by Mr. Crisp, rupture into the right auricle occurred in two, into the right ventricle in two also, and into the left ventricle once. In the forty cases of aneurism of this part of the artery noted by Dr. Swett, rupture into the right auricle occurred in two, and into the right ventricle in two also. In no case of aneurism of the transverse or descending portion of the arch, was rupture into the chambers of the heart observed.

Rupture into the *pulmonary artery* is rare ; out of 146 cases of aneurism of the ascending and transverse portions of the arch, in Mr. Crisp's table, it occurred but four times, and in the seventy-one noted by Dr. Swett, it occurred twice only. Rupture into the *superior cava* appears to be about as frequent, having occurred in four out the former list, and in two of the latter. Rupture in either of these situations did not occur in any case of aneurism of the descending thoracic aorta.

Rupture into the posterior mediastinum is limited to cases of aneurism of the transverse, or descending portion of the arch, or of the descending aorta. Rupture into the anterior mediastinum is not stated to have occurred in any case in the foregoing tables.

Rupture into the *pleura* is observed in aneurism arising from every part of the thoracic aorta, but is most common in aneurism of the descending thoracic portion ; thus, while in 146 cases of aneurism of the arch, in Mr. Crisp's table, and in seventy-one given by Dr. Swett, it occurred only eight times out of the former number, and six times out of the latter : it was observed in eight cases out of twenty-one, of aneurism of the descending thoracic aorta, in Mr. Crisp's table, and in five out of sixteen in Dr. Swett's list. Rupture into the left pleura is much more frequent than into the right, having occurred twenty times in the former, for nine in the latter. Rupture into the *lung* is less frequent than into the pleura, having occurred only eleven times in 254 cases.

\* On Diseases of the Chest.

Rupture into the *oesophagus* is most common in aneurism of the transverse and descending portions of the aorta, though it has also been observed in aneurism of the ascending portion of the arch. It occurred nine times in 175 cases in the first table, and in seven out of eighty-seven of the latter. Rupture into the *trachea* is, in a great measure, limited to aneurism of the transverse portion of the arch; and although the sac, in aneurism of this part of the artery, frequently contracts adhesion with the *trachea*, the patient seldom dies from rupture into this tube. Thus, in the 254 cases in the two series referred to above, rupture into the *trachea* occurred only thirteen times; while in twenty-one out of the 167 in Mr. Crisp's table, death which occurred without any rupture was apparently due to the pressure upon the *trachea* and bronchi. Rupture into either *bronchus* is less frequent than into the *trachea*; it occurred but eight times in the 254 cases. Rupture into the left *bronchus* is much more frequent than into the right; in six out of these eight cases it occurred on the left side; in the remaining two the side is not stated.

Rupture into the *vertebral canal* is extremely rare; no case of it is given in Mr. Crisp's table, and only one by Dr. Swett. *External rupture* compared with internal is very rare; it occurred only seven times in the 175 cases tabulated by Mr. Crisp, and only once in the eighty-seven cases given by Dr. Swett; while internal rupture occurred eighty-seven times in the former table, and fifty-eight times in the latter.

The process by which the contents of an aneurism escape, is not exactly the same upon a serous, as upon a mucous or cutaneous surface, as Mr. Hodgson first pointed out. When it reaches the cutaneous surface, a slough generally forms, the gradual separation of which is followed by hæmorrhage; the same occurs when the sac contracts adhesion with a canal lined by mucous membrane. When rupture occurs upon a serous surface, the membrane becomes gradually thinned, it cracks, and the contents of the sac escape. This I have observed to occur also upon the cutaneous surface.

When rupture occurs, it is not necessarily followed by an immediate fatal effusion of blood, the patient sometimes lives several hours, sometimes several days, or longer after it. Whether death is instantaneous or not, will depend upon the size of the aperture

and its situation, and in part upon whether the sac contains fibrin in its interior, or this is absent.

Rupture into the pericardium, unless the aperture is very small, or old adhesions had united the opposed layers of this membrane, is generally instantly fatal; the heart is suddenly compressed, and its action ceases. When rupture occurs into the pleura, or posterior mediastinum, the hæmorrhage is generally so profuse as to cause immediate death. When the aneurism bursts into the trachea or bronchi, a gush of blood from the mouth and nose generally indicates the accident, and the patient is usually quickly asphyxiated by the filling of the bronchial tubes. When rupture occurs into the pulmonary tissue, it is not necessarily immediately fatal. In a case recently under the writer's care, the patient experienced the sensation of something having given way internally, followed by intense dyspnœa, paleness of the surface, anxiety, and a presentiment that he could not live long; he rallied somewhat, however, but two days subsequently on getting into bed, he suddenly became weak, and died in a few minutes. On examination, the aneurism was found to have ruptured into the right lung, and then through it into the right pleura.

When the aneurism opens into the œsophagus, after the first gush of blood, the orifice may be plugged by a portion of fibrin from the interior of the sac, and the fatal result may be postponed for several days. Rupture into the superior cava or pulmonary artery is never instantly fatal. When the aneurism opens externally, death is seldom instantaneous; the hæmorrhage is checked by nature or by art, and the patient seldom dies until after several successive recurrences of the hæmorrhage. In a case under the writer's care, where the aneurism, after forming an external tumor, became diffused under the pectoral muscle, the patient survived the rupture fourteen days. In another, in which the spinal canal was exposed, the patient lived for upwards of a month after the supervention of paraplegia, and died then of rupture of the sac into the left pleura.

Rupture of aneurism of the aorta may occur suddenly and unexpectedly; or it may be preceded by increased restlessness, uneasiness, dyspnœa, and pain; sometimes by a sense of impending danger: a severe fit of coughing generally precedes rupture into the trachea, or into one of the large bronchi. When the patient

survives the first gush of blood, he generally tells us that he was sensible of something having given way internally. The rupture may occur on the patient making some slight muscular exertion, such as rising or turning in bed, or getting into bed; or it may occur when he is at perfect rest. In several instances, the patient had been conversing with the occupant of the next bed, and appeared to have fallen asleep; but on coming to him he was found to be dead. When rupture occurs at an earlier period of the disease, it usually follows some violent exertion, as lifting a heavy body, or in running, struggling, &c.

Although rupture is a frequent termination of thoracic aneurism, many cases terminate fatally without its occurrence; thus in the 167 cases tabulated by Mr. Crisp, rupture occurred in ninety-four, and sixty died without any rupture; and in the eighty-seven cases noted by Dr. Swett, rupture is stated to have occurred in fifty-nine, and thirty-two died without rupture. In aneurism of the ascending part of the arch, and of the descending thoracic aorta, death by rupture is much the more common termination; in aneurism of the transverse portion of the arch, death without rupture appears to be nearly as frequent as death from rupture.

When the disease terminates fatally without rupture of the sac, this may be due to asphyxia, to syncope, or to coma, according to the parts which are injuriously compressed; or the constant pain, dyspnoea, and want of sleep may wear away the thread of life, and the patient dies of exhaustion. This I have several times observed, when the sac had contracted adhesion with the trachea, compressing it; and the sufferings are usually severe and protracted in such cases.

The assigned causes of death, in the cases tabulated by Mr. Crisp, were in twenty-one, pressure on the trachea and bronchi; in eight, serous effusion into the pleura or pericardium; in three, suffocation; in two, syncope; in two, coma; in two, exhaustion; and in two, phthisis. In the remainder, death apparently resulted from pressure on the superior cava or pulmonary artery, from cerebral congestion, or apoplexy, pericarditis, bronchitis and hæmoptysis, pulmonary apoplexy, pressure on the recurrent nerve, dysentery, rupture of the ascending aorta, exhaustion and suffering, or external violence,

The period which intervenes between the development of tho-

thoracic aneurism, and the fatal termination, is very variable; I have known it to terminate within six weeks from the period the patient first applied for advice, and I have known it to last nearly as many years, and the patient ultimately to die of some other disease.

## CHAPTER XXV.

**SYMPTOMS OF THORACIC ANEURISM.—SYMPTOMS PREVIOUS TO THE ANEURISM FORMING AN EXTERNAL TUMOR.—SYMPTOMS WHEN THE ANEURISM FORMS A PULSATING TUMOR UPON THE SURFACE.**

THE symptoms of thoracic aneurism present great diversity and vary according to a number of circumstances, as the size of the aneurismal sac, and the direction which it takes, the part of the artery from which it springs, whether its increase in size has been slow or the contrary, whether it is concealed within the walls of the chest, or forms a tumor externally, and particularly, the nature of the parts which are compressed, stretched, displaced, or otherwise injured by the growth of the tumor. Indeed, a knowledge of the parts with which the thoracic aorta is anatomically related, constitutes in a measure a key to its symptoms. Thus, this artery is more or less intimately related with :

1. The pleuræ, lungs, trachea near its bifurcation, and the bronchi, particularly the left.

2. The trunk of the pulmonary artery, the right pulmonary artery, and the pulmonary veins.

3. The pericardium, and right and left auricles.

4. The superior vena cava, and the venæ innominatæ.

5. The arteria innominata, the left carotid, and left subclavian.

6. The œsophagus and thoracic duct.

7. The vagi nerves, the phrenic, and the recurrent on the left side.

8. The great cardiac plexus, the cardiac ganglion, and the right inferior cardiac nerve.

9. The bodies of the dorsal vertebræ, the sternum and ribs.

An aneurism springing from the thoracic aorta may thus :

1. By pressing upon the lungs, trachea, or upon either bronchus, give rise to cough, dyspnœa, stridor, croupy or wheezing respira-



tion. Or by compressing a single bronchus, it may prevent the entrance of air into the portion of lung supplied by it.

2. By compressing the œsophagus it will give rise to dysphagia, and interfere with nutrition.

3. By compressing the superior vena cava, or venæ innominatæ, it will interfere with the return of the venous blood to the right auricle.

4. By its pressure upon the left carotid or subclavian, or upon the arteria innominata it will diminish the calibre of these arteries, and eventually may cause obliteration of their canals.

5. By compressing the thoracic duct, it may prevent the entrance of chyle into the circulation.

6. By compressing the pulmonary artery, it will prevent the free passage of the venous blood to the lungs, and by compressing the pulmonary veins it will interfere with the return of the blood to the left side of the heart.

7. By pressing upon the heart it might interfere with its movements or cause displacement of the organ, protruding it forwards or downwards, according to the situation of the sac, and its size.

8. By compressing the left recurrent nerve, it may give rise to aphonia and hoarseness.

9. By compressing the vagi nerves the respiratory process may be interfered with, and congestion or œdema of the lungs induced.

10. By compressing the phrenic nerve upon the left side, the functions of the diaphragm may be interfered with, and spasm or dyspnœa excited.

11. By its pressure upon the great cardiac plexus, the cardiac ganglion, or the branches of the cardiac nerves, the functions of the heart may be more or less disturbed.

12. By acting upon the bodies of the dorsal vertebræ, or upon the sternum or ribs, erosion of these bones is produced. Finally, by the irritation excited in the sensitive branches of nerves in its vicinity, which it compresses or stretches, pain and various distressing sensations are occasioned.

We have seen that the parts with which the arch of the aorta, and the descending portion of the thoracic aorta are anatomically related, are very different; the organs, consequently, which are liable to be compressed, stretched, or otherwise injured by the growth of an aneurism in the one situation, are not the same as in

the other, and the train of symptoms attending each is different. In describing the individual symptoms, we might arrange them as they belong to the one or the other, and this would be a very convenient arrangement in a series of cases of the disease, but would lead to many repetitions here; I propose, therefore, to consider the symptoms under the two following heads:

1. Those which are presented during what may be termed the period of latency of the aneurismal tumor.

2. Those which are observed when the aneurism forms a pulsating tumor upon the surface.

Under the first head we have:

1. Stridor, and wheezing respiration.

2. Dysphagia.

3. Difference in the pulse at each wrist, or absence of the pulse in one radial artery, or in the temporal or carotid arteries of one side.

4. Hoarseness, or aphonia.

5. Dyspnoea and cough.

6. Pain.

7. Venous congestion of the neck.

8. Emaciation.

9. A pulsation deep in the chest, of which the patient may or may not be sensible.

10. A double sound on auscultation, distinct from that of the heart.

Under the second head we have, in addition to any of the preceding, or independent of them:

1. A pulsation visible to the eye and perceptible to the hand, at some part of the parietes of the chest.

2. Dulness on percussion over the site of the tumor, or pain on pressure, or percussion on this part.

3. A double impulse, and a double sound over the site of the tumor.

4. A murmur occasionally accompanying either the first or second aneurismal sound; very rarely both.

#### SYMPTOMS PREVIOUS TO THE ANEURISM FORMING AN EXTERNAL TUMOR.

*Stridor in respiration* has been recognized, from the time of Morgagni, as a sign of aneurism of the aorta compressing the trachea. It is observed whenever the sac contracts adhesion with

the trachea, and by its pressure causes flattening of the tube, or diminishes its calibre. It is a very characteristic symptom, is generally better marked during inspiration than expiration, and is occasionally loud enough to be heard at a distance from the patient. It is occasionally accompanied by dysphagia, the tumor compressing the oesophagus likewise; and as the pressure of an aneurism cannot diminish the calibre of the trachea without irritating its lining membrane, stridor is almost always accompanied by cough, which is hoarse and croupy. Stridor is most common in aneurism of the transverse portion of the arch of the aorta, and when the sac springs from the back of the artery, and a small aneurism in this situation will give rise to it. It is not, however, peculiar to aneurism, for any other tumor compressing this tube might produce a similar result.

Dr. Stokes lays much stress upon the stridor in these cases being from below, and states that from want of attention to this point, the affection has been mistaken for chronic disease of the larynx; but the sound is so peculiar, and so characteristic, that once heard it can scarcely be mistaken for any other; besides, the patient generally points to the trachea at the root of the neck, as the seat of his distress.

*Wheezing respiration* at an earlier period of the disease, and when the pressure upon the trachea is not sufficient to flatten this tube or to diminish its calibre, the respiration is wheezing, and accompanied by cough, which has the same characters. Wheezing respiration and cough may likewise be the result of pressure upon one of the bronchi, particularly the left, which, from its relation to the arch of the aorta, is very liable to suffer in aneurism springing from its transverse portion. The wheezing accompanies the inspiration particularly, and may be constant, or intermittent, or only audible on a full inspiration. The irritation occasioned by the pressure sometimes gives rise to the sensation of something being present in the air-tubes which the patient would be relieved by expectorating, and he makes many ineffectual efforts to do so.

In a few instances, the sides of one of the bronchi have been pressed together by the growth of an aneurism, and the entrance of air into the portion of lung supplied by it interrupted, accompanied by absence of the respiratory murmur, but without dulness or percussion in that part of the chest, with, perhaps, puerile res-

piration in the opposite side. This sign of an aneurism compressing a large bronchus, was first pointed out by Dr. Stokes.

*Dysphagia* is mentioned by Morgagni as a sign of thoracic aneurism, and is observed whenever the aneurismal sac contracts adhesion with the œsophagus, and compresses it; and according to the amount of pressure the swallowing of solids may be difficult, or impossible. It is most liable to occur in aneurism of the transverse portion of the arch, and when the sac springs from the posterior part of the vessel. It is not as common a symptom as the preceding, and although a valuable sign when present, and when combined with others, it is by no means pathognomonic of aneurism, because any other tumor compressing the œsophagus would give rise to it, and it is a prominent symptom in stricture of this tube.

The site of the dysphagia is sometimes referred to the top of the sternum, sometimes to the epigastrium, or it may be referred to any intermediate point between; the patient has sometimes the sensation that the pressure is at the back of the œsophagus; it is not constantly present to the same degree, varying in the same subject at different periods, owing probably to the varying state of distension of the sac. In some instances it is temporary, and appears to be due more to spasm than to diminution of the calibre of the œsophagus. Even when dysphagia has been present for a time in a marked form, and is undoubtedly due to the pressure of an aneurism, it may subside considerably or disappear completely; a case in which this occurred was recently under the writer's care; it resulted probably from the aneurismal sac having bulged out laterally, by which the direct pressure upon the œsophagus was taken off.

Flatulent distension of the stomach is occasionally a very troublesome symptom in cases of this kind, particularly when the pressure on the œsophagus is such, that the patient is reduced to the necessity of living on liquid nutriment; and there is then sometimes great difficulty in eructation, causing great distress to the patient.

*Difference in the pulse at each wrist.*—The radial pulse not unfrequently differs in strength and fulness on each side, being either more feeble, or being nearly or altogether imperceptible in one radial artery, while it preserves its normal strength in the

other. This is observed, when the subclavian upon the left side, or the innominate upon the right is compressed by the growth of an aneurism springing from the arch of the aorta. It is more frequently observed upon the left than the right side, owing to the long course of the left subclavian in the thorax. The irritation occasioned by the pressure causes exudation of lymph in the interior of the artery, and it eventually becomes obliterated at the point, much in the same way as when a ligature is applied.

It sometimes happens that the carotid upon the left side is compressed along with the subclavian, and the pulse may be absent in the temporal, or carotid on that side. This is more frequently observed, however, on the right side owing to compression of the arteria innominate. In a case in which this occurred, where the radial pulse was very indistinct, and no pulse was to be felt in the right carotid, or temporal, the right side of the neck in the course of the carotid artery was painful to the touch, and the patient complained of numbness of the arm. Difference in the strength of the pulse at each wrist, or absence of the pulse in one radial, or carotid artery, is a valuable sign of aneurism of the arch of the aorta, when combined with others, but it is also absent in many cases. It is not observed in aneurism of the descending thoracic aorta.

*Hoarseness or aphonia*, the voice becoming husky, or being reduced to a mere whisper, is an occasional symptom of thoracic aneurism, particularly when the sac springs from the left side of the transverse portion of the arch, and the *recurrent nerve* on that side is involved in the tumor, and stretched or compressed. We know that the muscles attached to the arytenoid cartilages receive their motor filaments from the recurrent nerves, and experiments upon animals, have shown that the division of both these nerves is followed by complete loss of voice, owing to paralysis of the muscles which move the arytenoid cartilages, while the division of a single nerve diminishes the voice.

Pressure upon this nerve by an aneurism of the arch of the aorta may be combined with pressure upon the left bronchus, or trachea, when more or less stridor, or wheezing respiration will accompany the aphonia. In a few instances, the irritation caused by the simultaneous pressure upon these two parts, has been such as to give rise to symptoms simulating laryngitis, or spasm of the glottis.

*Dyspnœa* has been long recognised as a symptom of thoracic aneurism; it is, however, a variable one, being little urgent in some cases, and the most prominently distressing symptom in others. In aneurism of the descending thoracic aorta, in which pain is the most prominent symptom, dyspnœa is often absent altogether; in aneurism of the ascending portion of the arch, cough is often more troublesome than dyspnœa; it is in aneurism of the transverse portion of the arch that it is generally most marked, and most distressing. It may be due to pressure upon the trachea or bronchi, to compression of the lung, or to the compression or stretching of the par-vagus, phrenic, or recurrent nerve upon the left side, or to two of these causes combined.

Compression of the pulmonary tissue, by the growth of an aneurism, in general takes place so gradually that the organ has time to adapt itself to the change; this, therefore, is seldom more than a secondary cause of dyspnœa.

When the dyspnœa depends on pressure upon the trachea, it is accompanied by stridor or wheezing respiration, and often amounts to orthopnœa; the patient being incapable of assuming the horizontal posture, in bed he lies upon his face, or he sits up, or leans forward constantly, in which position only perhaps, he obtains a little sleep. The dyspnœa is constant, though it likewise occurs in paroxysms—in these, the face becomes congested and livid, sometimes almost black, and the patient appears to be in imminent danger of suffocation, the urine and fœces being in extreme cases discharged involuntarily. A paroxysm may be induced by very trifling exertions, such as getting into or out of bed, by mental emotion, sometimes by a fit of coughing, or by speaking; or it supervenes when the patient has been asleep for a few minutes. In such cases, there is often an accumulation of mucus in the trachea, which the patient finds great difficulty in expectorating.

Dyspnœa may, however, be present, although neither the trachea, bronchi, or lungs are compressed by the tumor. In such cases, it may have its cause in irritation or injury to the par-vagus or phrenic nerve on the left side.

The *vagi* nerves are intimately associated with the function of respiration, and experiments upon animals have shown, that after division or ligature of both these nerves, the respiration became gradually slower and more difficult, the temperature of the animal

fell, the lungs became congested, and a bloody serum exuded into the minute bronchi and air-cells. Division of the vagus nerve on one side in animals, is said to cause no impediment to respiration; but even if this were so, it does not follow, that if the vagus nerve on the left side, or its pulmonary branches, are stretched or compressed by the growth of an aneurism, the functions of the lung on that side may not be seriously disturbed.

The *phrenic* nerve upon the left side is likewise so situated that a large aneurism springing from the arch of the aorta, may irritate, compress, or stretch it, and as a result, existing dyspnoea may be aggravated, or spasmodic attacks of severe dyspnoea induced, with pain referred to the site of the diaphragm. Mr. Burns\* mentions a case where a painful sense of stricture about the diaphragm, was "probably occasioned by the aneurismal sac irritating the phrenic nerve;" and he adds that Dr. Simmons had seen the same symptom in three other cases.

*Cough.*—Whenever dyspnoea is a prominent symptom, cough is generally associated with it, and this is always the case when the aneurismal sac presses upon the trachea, or large bronchi, or when the pulmonary tissue is compressed and condensed. In the former case, the cough which usually occurs in paroxysms is raucous and croupy, accompanied by stridor or wheezing respiration, and it is usually aggravated by the recumbent posture. In other cases, the cough presents nothing peculiar. The expectoration is watery and frothy, or scanty and difficult, accompanied in cases of pressure on the trachea or bronchi, by the sensation of a foreign substance in the tube. In advanced cases, the expectoration is often streaked with blood, or it is rust-coloured, or it may present the prune-juice character, particularly if the pulmonary tissue is much compressed by the growth of the tumor.

*Pain* is a frequent, and sometimes a very urgent symptom in aneurism of the aorta, whether of its thoracic or abdominal portions. It is not unfrequently the earliest symptom which attracts the patient's attention, or which leads to a suspicion of the existence of the disease; and in a few instances it is the only one from which he suffers all through.

It is only within a comparatively recent period, that the im-

\* On Diseases of the Heart, p. 252.



portance of pain, as a sign of aortal aneurism, has been recognised, and its value as an aid to diagnosis fully appreciated. We are indebted to Dr. Thomas Beatty\* for first calling attention to it in abdominal aneurism; Dr. Greenet† subsequently pointed out the frequency, and the obstinate nature of the pain in thoracic aneurism; Dr. Law‡ was, however, the first to describe its twofold character, and to dwell particularly upon its importance as a diagnostic sign before any external tumor forms, or before other symptoms arise to attract attention. I do not, however, mean to say, that this symptom had been overlooked previously—all writers, from Morgagni down, allude to it, and Laennec§ particularly mentions it. “In many instances, where the aneurism occupied the descending portion of the aorta, and where the bony matter of the vertebral column was acted on, the patient complained (he observes) of a severe, gnawing pain, referred to the corresponding part of the back or loins, which he compared to the action of a boring instrument; in others the pain resembled that of rheumatism, and in some instances it had all the characters of neuralgia.” He adds that he was led to suspect the presence of aneurism, in one case, from the seat and character of the pain, and from the resonance on percussion being diminished at the part.

The nature of the pain in aortal aneurism is peculiar; it has generally, as Dr. Law first pointed out, a twofold character, viz.:

1. A dull, aching pain, which is constantly present.
2. A sharp, lancinating pain, which is intermittent, or only experienced at intervals.

The dull, aching pain, in thoracic aneurism, is felt in the region of the cervical or dorsal spine, between the scapulæ, or in the scapular region on either side, in the right or left shoulder, arm, or side of the neck, or in the sternal or mammary regions. The lancinating pain is felt in the same situations, or it extends along the ribs to the left shoulder and arm, or it shoots through the chest from the præcordial to the left scapular region. Sometimes, however, the patient makes no distinction between the two kinds of pain, but describes it as always intensely boring, or burning. A remarkable peculiarity in the pain, which I have noticed so frequently as to regard it as almost pathognomonic, is that it is

\* Dub. Hosp. Reports, vol. v.

† Dub. Jour. of Med.

‡ Dub. Jour. of Med.

§ Auscultation Mediate, t. ii.



much more severe at night, than during the day, it even sometimes comes on regularly at a certain hour, and subsides at a fixed hour in the morning. The weather seems sometimes to have an influence upon the pain: a patient in hospital last winter always suffered most in frosty weather, and said he knew when frost was coming by the increase of pain.

The constant, dull pain has often the characters of rheumatism or neuralgia, and is frequently considered to be such by the patient and prescribed for as such by his medical attendant. The lancinating, intermittent pain presents many of the characters of angina. When the two kinds of pain are present at the same time, the sufferings of the patient are greater than in most diseases.

When thoracic aneurism approaches the surface, or forms a tumor externally, pain or soreness of the integuments covering it, is often, in addition, complained of, which is increased by pressure; indeed, sometimes even the pressure of the stethoscope upon the part can scarcely be borne.

If aneurism of the aorta was always attended by pain, such as has been described, and if it always had the same characters, no difficulty would ever be experienced in the diagnosis. This, however, is far from being the case, sometimes little complaint of pain is made from beginning to end, at others it has those vague characters which are not calculated to attract attention. But, these circumstances, which appear to militate against its value as a sign of aneurism, in reality increase it, because pain of a severe and obstinate nature is the earliest symptom in precisely those cases, where the aneurism would otherwise remain longest unsuspected and undiscovered, owing to the absence of almost every other sign, and to the length of time which elapses before a tumor appears externally. Thus, when the aneurismal sac springs from the descending portion of the thoracic aorta, it cannot compress any of the important organs or parts, which an aneurism springing from the arch does; but it can scarcely form here without coming in contact with the bodies of the dorsal vertebræ, with which it contracts adhesion, followed by erosion of the bone and severe pain; and this appears to be almost the only direct sign of aneurism of this part of the vessel, unless the sac attains such a size as to form a tumor externally. The pain in these cases is often referred to a space which could be covered by a crown piece, a little above the

inferior angle of the left scapula, as well as to the left nipple in front, and it shoots from the one point to the other, and occasionally extends to the left shoulder or arm.

*Cause of the pain.*—With respect to the cause of the pain, pathologists are not agreed; this seems to have arisen in some measure, from their seeking to refer it to some one cause, to the exclusion of others. But as the parts, with which an aneurismal sac comes in contact, are so different, as the sensibility of these parts varies so much, and as the amount of injury they suffer from the pressure differs likewise, the cause of the pain cannot always be the same, and it ought to vary in character and in severity in different cases, which we know it does.

The cases of thoracic aneurism, in which pain is by many degrees the most prominent, constant, and distressing symptom, are those in which the sac springs from the descending portion of the thoracic aorta, and contracts adhesion with the bodies of the dorsal vertebræ, ending in erosion of these bones, and of the ribs near their articulations. Here the pain must be due, in part at least, to the destruction of the periosteum and bone; in fact it resembles the pain in ulceration of the cartilages of the joints extending to the bone; like the latter it increases at night, and remits during the day, and it is severe at points remote from the seat of the disease. But as an aneurism in this situation, cannot act upon these parts without irritating and compressing the thoracic ganglia of the sympathetic, which pass down over the heads of the ribs, and the branches which come off from them, the pain may be partly due to this also.

In aneurism springing from the arch of the aorta, the pain, which is much less intense, appears to be due, partly to the irritation of the sensitive branches of nerves in the vicinity of the sac; and partly to the compression and stretching of the tissues with which the aneurismal sac, in enlarging, comes in contact; which we know is sufficient to cause exudation of lymph, and adhesion between these parts and the sac, and to interfere materially with their functions.

*Venous congestion.*—The ascending portion of the arch of the aorta is so situated, that an aneurism springing from it may readily compress the superior cava, while aneurism of the arch may compress the vena innominata on either side, the effect of

such pressure will be to impede the return of the venous blood from the head, face, neck, and upper extremities, to the right side of the heart; the jugular veins become distended, tortuous, and present almost a varicose condition; the neck is swollen, the hollows naturally existing at its root are obliterated, and the tippet-like appearance of this part, described by Dr. Stokes, is occasionally present. Although distension of the jugular veins is frequent, visible pulsation in these vessels is rare. The superficial veins at the upper part of the chest are occasionally larger, and more prominent than natural on the side on which the aneurism is seated, and I have observed distinct pulsation in an enlarged vein in this situation.

If the sides of the superior cava are pressed together, and its channel is obliterated, it may give rise to anasarca of the upper half of the body, or this part may suddenly assume a deep blue colour. Thus in a case recorded by Dr. Law, the patient, a man about forty years of age, suddenly felt a fulness in the face, accompanied by great oppression in the præcordial region; the face, neck, hands, and arms then assumed a dark livid hue (as deep as occurs in Asiatic cholera); the face, neck, and upper part of the chest were likewise much swollen, the eyes were deeply suffused, and there was considerable serous infiltration into the subconjunctival cellular tissue. The patient died a few days afterwards, and on examination an aneurism of the ascending portion of the arch of the aorta was found, which had pressed the sides of the superior vena cava together.

In another recorded by Dr. Watson, the patient's face, neck, and arms were tumid and anasarcous to an enormous degree; while there was not the least trace of swelling or œdema below the ribs. "He looked as if his upper half had been stuffed, and except that it was distressing, his appearance was extremely comical." The countenance was livid, the eyes seemed starting from their sockets, the cellular tissue beneath the conjunctiva was cedematous, and his arms were so swollen that they projected from his sides. The epigastric veins were visible and tortuous, and a free communication by anastomosis existed between these and the mammary veins. After the patient's death, a large aneurism of the aorta was found, which had compressed the descending vena cava, and this vessel was quite impervious a little above the right auricle.

When the aneurism has contracted adhesion with the superior cava, the pressure may give rise to interstitial absorption of the coats of the vein, and this may go on until the sac opens into the vein, when a spontaneous varicose aneurism is formed. This is a very rare termination, and the patient does not live long after its occurrence. An interesting case of it has been recently recorded by Dr. Mayne.

The patient, a woman fifty years of age, when stooping suddenly felt as if strangled, the face became livid, and the breathing embarrassed, with a sensation of giddiness and suffocation. When seen by Dr. Mayne, the whole upper part of the trunk was of a deep plum-colour, while the lower portion and the abdominal extremities were pale, the eyes were protruded from their orbits, and the face, neck, and upper portions of the chest were swollen and puffy; all the veins of these parts were enormously distended; a single heaving systolic impulse was felt, with a distinct fremitus, and a loud whizzing murmur was audible over all the chest. On examination after death, a large aneurism was found, between which and the superior vena cava, a communication had been established. The aperture resembled a button hole in shape, with sharp irregular edges.

*Compression of thoracic duct.*—The thoracic duct, as it ascends towards the neck, behind the arch of the aorta, and left subclavian artery, might be compressed by an aneurism springing from the posterior surface of the transverse portion of the arch at the left side, or from the descending portion of the arch, followed by enlargement of the absorbents and lymphatic glands, and death from inanition; but it is extremely rare; Laennec, however, has recorded a case;\* the aneurism sprung from the descending portion of the arch.

*Displacement of the heart.*—Notwithstanding the large size which aneurism of the arch of the aorta often attains, and its proximity to the heart, this organ is very seldom displaced. This is owing to the usual direction of the sac being upwards, forwards, or backwards; and, as it increases in size, it contracts adhesion with the parts it comes in contact with, by which pressure upon the heart sufficient to displace the organ downwards, or to the right or left side is prevented.

\* Journal de Médecine, t. xii.

When an aneurism forms in the *descending thoracic aorta* immediately behind the heart and the tumor is large, the heart may be protruded forward by it, causing a stronger and more diffused impulse, and simulating hypertrophy with dilatation of the ventricles. A case in which this occurred was under the writer's care some years ago—any other tumor in the same situation might, however, give rise to the same phenomenon, and in each there would be dulness on percussion posteriorly; they would be distinguished by the amount and kind of pain the patient experienced, and by a double sound resembling that of the heart being audible, in the case of aneurism, posteriorly at the site of the dulness. If the patient lives long enough, the diagnosis is generally cleared up by the aneurism making its way to the surface, and forming a tumor posteriorly, or by its rupture internally.

*Emaciation.*—When thoracic aneurism compresses the œsophagus, and prevents a sufficient amount of nutriment being taken, the patient, as might be expected, gradually emaciates. Emaciation to a greater or less extent is, however, generally present in the disease when it has made much progress, whether the œsophagus is compressed or not, and in some instances this is a very marked symptom. In two cases, recently under the writer's care, the patients stated that they each had lost two stone in weight within a comparatively short period. When it is not due to pressure on the œsophagus interfering with nutrition, it is generally the result of the long continued pain, want of rest, dyspnoea, and the harassing cough; and it is commonly present when the aneurism compresses the trachea near its bifurcation.

*Anasarca* of the lower extremities is occasionally present in the advanced stage of thoracic aneurism when the sac has attained a large size; it is not, however, a sign of any importance, because it never occurs until other and more positive signs of the existence of aneurism have preceded it. *Anasarca* of the face and upper extremities has been already alluded to in noticing the symptoms of compression of the superior cava; and when the vena innominata or the subclavian on either side suffer compression, anasarca of the arm and face on that side usually supervenes.

*Paraplegia* has supervened in a few instances, where erosion of the bodies of the dorsal vertebræ had gone on until the vertebral canal was exposed. This is most liable to occur in aneurism

of the descending thoracic aorta. In a case under the writer's care, the paraplegia commenced in the left lower extremity, and a week subsequently extended to the right, sensation was not abolished, but the patient suffered much from spasmodic twitchings in the paralysed limbs, and they were with difficulty kept warm; he had occasionally some difficulty in passing urine, but he never required the use of the catheter. The patient lived upwards of a month in this state, and death was then the result of rupture of the aneurism into the left pleura, although it had formed a large tumor at the back of the chest on the left side. On examination, the finger could be passed from the aneurismal sac into the vertebral canal, the edges of the aperture in the bone were rounded off, and neither rough or jagged.

Lastly, in a few instances on record, the only symptoms noticed were flatulence, and a sensation of something rising in the throat, resembling the globus hystericus, which masked the more serious disease, and probably depended upon irritation of the pneumogastric nerve.

*Percussion* is not of much assistance to the diagnosis as long as the aneurism is small, and seated deep in the chest, particularly when the latter is capacious. When it increases in size, a dull sound will be yielded by percussion over its site; and if it approaches the surface, or makes its way through the ribs or sternum, even the slightest percussion is sometimes so painful that it cannot be borne.

*Pulsation or sound in abnormal situations.*—Although the aneurism is buried, and concealed within the bony walls of the chest, we can often detect pulsation or sound in some abnormal situation. Thus, sometimes by pressing the finger behind the first bone of the sternum, at the root of the neck, we are able to detect a pulsation; at others, and particularly when the chest is narrow, if we lay one hand upon the back of the chest, and the other upon the front, and compress it between them, a pulsation deep in the chest may be perceived. Whenever this sensation is communicated to the hand, it will become more distinct if we apply the stethoscope opposite to the point at which it is perceived, and we will then generally hear a double sound, resembling that of the heart, but distinct from it. These signs become more distinct the nearer the aneurism approaches the surface.

## SYMPTOMS OF ANEURISM FORMING AN EXTERNAL TUMOR.

When the aneurism reaches the surface, or forms a pulsating tumor externally, in addition to any of the symptoms which have been enumerated, or independent of them, a certain number of new signs become evident, which are more certainly diagnostic, and were formerly regarded as the only positive signs of the existence of thoracic aneurism. These are afforded principally by manual and physical examination of the chest.

An aneurism which forms a pulsating tumor externally at an early period, is usually accompanied by fewer of the distressing symptoms which have been enumerated, than when it had remained long buried in the chest, and eventually reached the surface; because in the latter case, it is certain to have contracted adhesion with some of the parts with which it came in contact; while in the former, it had probably contracted adhesion with none.

The size of the tumor varies considerably, it may be scarcely elevated above the surface, or it may attain the magnitude of the foetal head. When the tumor rises up in the neck it occasionally attains a large size: I have seen it reach the chin, under these circumstances. When the aneurism springs from that part of the aorta within the sac of the pericardium, it usually ruptures before forming a tumor externally.

The *strength of the impulse* varies much in different cases, being sometimes inconsiderable, sometimes powerful and heaving, agitating the whole frame, and causing great distress and inconvenience. When the impulse is slight, it may be owing to the sac having contracted deep adhesions, or to its being partly filled with fibrin, or to its being bound down by fascia. When the impulse is powerful, it may be owing to the thinness of the coverings of the aneurism, or to a hypertrophied left ventricle propelling the blood with increased force; sometimes to temporary increased irritability of the heart.

The impulse of aneurism of the thoracic aorta differs from that of aneurism in other situations, in being *double*, not single; the first impulse coinciding with the ventricular systole, the second with the ventricular diastole. Even before the aneurism has reached the surface, the double impulse is generally perceptible to



the ear laid on the stethoscope, although no shock may be felt by the hand applied to the part.

The double impulse of thoracic aneurism is closely related to its double sound, the first impulse corresponding with the first aneurismal sound, the second with the second aneurismal sound. The mechanism of its production is similar to that of the double sound, and will be considered when we come to speak of the latter.

#### SOUNDS HEARD ON AUSCULTATION.

It was laid down by Laennec, that a loud, single bruit de soufflet, synchronous with the first sound of the heart, is characteristic of aneurism of the thoracic aorta; this, however, is far from being the case, in many instances no bruit is audible from first to last, and in every case which I have examined, the sound was *double*, not single.

I am not aware by whom the occurrence of a double sound, and double impulse in thoracic aneurism was first noticed. Dr. Stokes, to whom it has been attributed, writing in the year 1834, (in commenting upon "a case of aneurism of the arteria innominata") observes: "This double pulsation of aneurismal tumors has been already noticed by several writers, and by some has been thought to arise from the contact of the aneurismal tumor with the heart itself, by which it receives its double impulse, an explanation evidently inapplicable to the present case."

The *double sound* of thoracic aneurism, which may be termed its *normal double sound*, bears often a close resemblance to the double sound of the heart, in some cases so much so, that if the eyes were shut, it could with difficulty be distinguished. Indeed, the remarkable similarity between the second sound of thoracic aneurism, and the second sound of the heart is shown by the manner in which almost every writer has sought to explain it—viz., by its being conveyed to the aneurismal tumor from the heart, owing to its proximity to this organ; but as the sound diminishes in intensity, or may disappear altogether between the sac and the heart, this explanation cannot be admitted. The normal double sound of thoracic aneurism, not only resembles the normal double sound of the heart in its sensible characters, but it further resembles it in one or other or both the aneurismal sounds being some-

\* Dub. Jour. of Medicine, July, 1834.



times converted into, or replaced by a murmur; thus sometimes the first sound is converted into a murmur; occasionally a murmur is heard only at the period of the second sound; and in a few instances both sounds are morbid.

#### MECHANISM OF PRODUCTION OF THE DOUBLE SOUND AND IMPULSE.

The explanation of the mechanism, by which the double sound and double impulse in thoracic aneurism are produced, appears to me to be simple. In seeking to explain it, I must premise:

1. That the sac of a thoracic aneurism possesses no elasticity, because, 1st it is in a great measure formed by the cellular coat of the artery, which possesses none. 2ndly, it almost always contracts adhesion with the parts it compresses, which if it did possess elasticity, would prevent this property from coming into operation. 3rdly, its exterior is often coated with lymph, the result of the irritation its pressure excites in neighbouring parts, by which it is thickened and strengthened. 4thly, its interior is often lined with fibrin in concentric laminæ, which adhere to the interior of the sac, and would prevent it from collapsing even if it contained no blood.

2. The orifice by which the sac and the artery communicate, is a permanently open one, and while it permits of the entrance of the blood during the ventricular systole, it cannot prevent regurgitation when the ventricular systole ceases, if the sac is so situated, that this fluid by its own gravity can regurgitate into it.

3. An aneurismal sac in any part of the body contains always more blood during the period of the ventricular systole, than during the diastole, shown by its being more distended in the former than the latter period of the heart's action.

*First impulse and sound.*—When the ventricular systole ensues, the blood transmitted by the left ventricle entering the aneurismal sac displaces a portion of that which it previously contained, and as the sac has but one orifice, there must be a current into and out of the sac, through the same orifice, at the same moment, the blood which enters, displacing a portion of that previously contained in it. The sudden distension of the sac which occurs at the moment, is the cause of the first impulse; and the friction of the blood against the parietes of the orifice of the aneurismal sac generates sound, which constitutes the *normal first sound* of aneu-

rism. If the friction between the blood and the orifice of the sac is increased from any cause, a murmur will be developed, which will, of course, replace the normal first sound, because it is nothing more than this sound exaggerated. The murmur commonly heard is the bruit de soufflet, and whether it is present or absent, will depend upon the size and shape of the orifice by which the sac and artery communicate, the size of the sac, and the direction which it takes, the force with which the blood is transmitted by the left ventricle, and whether this fluid is attenuated or the contrary.

*Second sound and impulse.*—The explanation of the mechanism by which the second aneurismal sound and impulse are produced, appears to me to be nearly as simple as that of the first sound and impulse.

I have said that the sac of an aneurism, in any part of the body, contains less blood during the period of the ventricular diastole than of the systole; it is then, in fact, partially unfilled compared with its previous state of distension. When the ventricular systole is over, and the vis a tergo ceases, the sac being comparatively unfilled, and its orifice a permanently open one, the blood from the aorta, and, in some instances, from the large arteries which arise from the arch, regurgitates into the sac simply by its own gravity to occupy the vacant space, just as regurgitation through permanently patent aortic valves occurs at the same period of the heart's action. This backward current of blood into the aneurismal sac produces sufficient friction to generate sound, and this constitutes the *normal second* aneurismal sound. If the force with which the blood regurgitates is considerable, the friction will be increased, and a murmur may be generated, which will take the place of the second sound; but as the force with which the blood regurgitates into the sac is always less than that of the systolic current from the ventricle, a murmur ought to be very rare with the second aneurismal sound, though not with the first, which we know to be the case.

The mechanism by which the second impulse is produced is quite analogous to that which gives rise to the second sound; the sac becomes partially redistended, owing to the sudden entrance of a small quantity of blood, and as the force with which the blood re-enters the sac is much less than that with which it is impelled into it by the ventricular systole, the second impulse ought to be

more feeble than the first, and ought, in fact, to amount simply to a slight shock, which it always is.

It is only in thoracic aneurism that we hear a second sound, or that we can detect a second impulse, and it is in aneurism of the arch of the aorta that these phenomena are best marked, particularly when the sac springs from the inferior, the anterior, or the posterior aspect of the artery in this part of its course. In aneurism of the abdominal aorta we have neither a double sound, except under some very rare circumstances, nor is a double impulse ever felt, because a sufficient amount of blood does not regurgitate into the sac to generate a second sound, or to communicate a second impulse. For instance, in aneurism of this vessel, the blood obviously cannot regurgitate from the *distal* side against gravity; while upon the cardiac side its own gravity, for the same reason, will impel the greater portion of it into the continuation of the artery, not into the sac; too little blood, therefore, can enter it either to generate sound, or to develop an impulse. Hence, we hear neither a second sound, nor feel a second impulse in aneurism of this part of the artery, or of any of its branches.

*Contraction of the pupil* upon the side of the aneurism, when the latter rises up in the neck, has recently attracted some attention as a sign of thoracic aneurism. It was first noticed by Dr. Gairdner,\* and subsequently by Dr. Banks;† it is supposed to be due to pressure upon the sympathetic nerve in the neck, experiments having shown that division of the sympathetic nerve in the neck in some animals is followed by contraction of the pupil on that side. More recently, M. Biffi found that division of the ascending or carotid branches of the sympathetic was followed by contraction of the pupil, though to a less extent than when the sympathetic itself was divided. He found also that irritation of these branches was followed by slight dilatation of the pupil.

I have not met with contraction of the pupil in any case of aneurism of this vessel; one subject, however, presented *dilatation* of the right pupil, on which side an aneurism subsequently rose up in the neck and formed an immense tumor reaching to the chin. The sight of the eye was perfect, and the patient stated that the dilatation had been noticed two years previous to any symptom of aneurism showing itself.

\* Ed. Jour., Aug. 1855.

† Dub. Hosp. Gaz.

*Dislocation of sternal extremity of clavicle.*—It occasionally happens that a gradual displacement upwards of the sternal extremity of the clavicle takes place when an aneurism rises up towards the neck; it is, however, rare, I have observed it only on the right side; the patient was a female, and the progress of the aneurism had been slow.

*Posture of the patient.*—The posture which a patient labouring under thoracic aneurism habitually assumes, has relation generally to the degree of dyspnœa present, or to the amount of pain experienced, which, of course, depends upon the size, position, &c. of the aneurismal tumor. In the early stage the patient can assume any posture he pleases; when the disease is advanced, lying upon the right side sometimes occasions a dragging sensation, at others he may be able to rest only on this side or inclined towards it, although the aneurism points at the left side. In most instances he lies inclined towards the side on which the aneurism presents.

When the aneurism presses upon the trachea, the subject of it is generally unable to rest upon his back, though he may lying upon his face, but he cannot remain for any time in the horizontal posture. In its advanced stages he is obliged to sit up constantly, or to lean forward, in which position only he obtains a little sleep. The patient is often restless and uneasy, continually changing his posture; and this is almost invariably observed for a short time previous to rupture of the sac.

When the aneurism has caused erosion of the bodies of the dorsal vertebræ, the patient may be unable to assume the erect posture without support. In a case of this kind, under the writer's care, where the vertebral canal was ultimately opened, the patient could not stand erect, the position which he assumed when out of bed was stooping forward, grasping the front of both thighs with his hands.

## CHAPTER XXVI.

### DIAGNOSIS OF THORACIC ANEURISM.—TREATMENT OF THORACIC ANEURISM.— PALLIATIVE TREATMENT.—CURATIVE TREATMENT.

#### DIAGNOSIS OF THORACIC ANEURISM.

THE difficulty of the diagnosis of thoracic aneurism has been a frequent theme of writers; and not a little that has been published has tended rather to increase than diminish this difficulty; owing partly to writers upon the disease taking too limited and contracted a view of the subject, as if one symptom or one set of symptoms belonged exclusively to aneurism in this situation; and partly to the erroneous ideas of previous authors, which have been adopted without proof, and propagated without consideration by succeeding writers; such as that an aneurism of this artery is always accompanied by a loud, single bruit de soufflet; or, that the arbitrary divisions of aneurism which systematic writers have made are characterised by distinct symptoms. The fact being, that bruit de soufflet, instead of constituting a constant auscultatory sign, is never heard in the majority of cases; and, instead of a single sound being characteristic of thoracic aneurism, a double sound is the rule, and a single sound the exception; while, whether the aneurismal sac is formed by all the coats of the artery, or by the external alone, after rupture of the internal and middle, will make no difference in the symptoms. The symptoms of aneurism of this vessel are not the result of the changes which the arterial tissues undergo; but they are the result of the compression, distension, stretching, and displacement of important organs or parts, or of the disturbance of function of vital organs in the vicinity of the aneurismal sac; and the growth of any other tumor in the same situation would give rise to pretty nearly the same symptoms.

The facility or the difficulty of the diagnosis must, it is obvious, be influenced by a variety of circumstances, particularly the size

of the aneurismal sac, its situation, the direction it takes, the capacity of the patient's chest, and the nature of the parts which are compressed. It is obvious, likewise, that aneurism in its early stage, may present quite a different train of symptoms from those of its advanced stage; and the symptoms, as we have seen, vary likewise as the aneurism forms a tumor externally, or remains concealed within the walls of the chest. Indeed the symptoms present such a remarkable contrast in different subjects, that the disease has been confounded with or mistaken for a variety of others. Thus, in some instances pain was the only prominent symptom from beginning to end, and the disease was set down as chronic rheumatism or neuralgia; sometimes, cough and dyspnoea were the only symptoms complained of, and the disease was referred to the lungs or bronchial tubes; sometimes difficulty of swallowing was the first circumstance which attracted the patient's attention, and he was supposed to be the subject of stricture of the oesophagus; sometimes hoarseness, loss of voice, stridulous or croupy breathing, were the most prominent symptoms, and the disease was supposed to have its seat in the larynx; at other times an unusual pulsation upon some part of the chest was the first circumstance which attracted the patient's attention; at others, a profuse hæmoptysis was the first symptom which alarmed him, and led him to seek advice; while sometimes, but more rarely, no single symptom from first to last, sufficient to attract the patient's attention, was present, and his sudden death led alone to the discovery of the disease.

When an aneurism of the thoracic aorta presents itself externally as a pulsating tumor, the diagnosis is of course easy; indeed, up to a comparatively recent period, this was supposed to be the only positive sign of its existence. But when the tumor neither approaches the parietes of the chest, nor compresses any important organ or part contained in this cavity, the diagnosis is attended with more difficulty. In some instances it will be facilitated by the mode of reasoning which the French term "par voie d'exclusion;" for example, if a patient exhibits one or more symptoms of thoracic aneurism, which may be common to it and to other diseases of the chest, but if we cannot refer them to any disease of the heart or lungs, the existence of aneurism is probable, provided the patient is a male, and his age that at which aneurism is most

frequent. In other cases, the patient's previous history may throw some light upon the diagnosis; thus, if a man who had previously laboured under spontaneous aneurism in one extremity, presents any symptom of thoracic aneurism, the probability of aneurism is strong. Or, if a man who has received a severe injury of the chest, or a violent strain, exhibits some time afterwards signs of pressure upon the trachea, large bronchial tubes, œsophagus, or any other part contained in the thorax, the existence of aneurism is probable; while if the previous habits of the patient had been such as to predispose to disease of the central organ of the circulation, it becomes still stronger.

In the very early stage of thoracic aneurism, the most carefully conducted examination may fail in detecting the disease; yet a few months later, perhaps, when it has made some progress, the diagnosis becomes comparatively easy. On the other hand, it may happen that the signs of aneurism are at one time so well marked as to leave little doubt as to the nature of the disease, yet on coming to examine the patient at a subsequent, and perhaps not very remote period, these signs may have disappeared, or they have been modified, or given place to others in consequence of another disease, as phthisis, having been set up in the interval.

Aneurism of the arch of the aorta, as a general rule, is more readily diagnosed than aneurism of the descending thoracic aorta, because from the anatomical relations of the arch, an aneurism springing from it must soon come to press upon some of the important parts in its vicinity.

Any other morbid growth in the same situation, by compressing the organs contained in the cavity of the thorax, would, however, give rise to nearly the same symptoms. Thus, a mass of enlarged bronchial glands seated at the bifurcation of the trachea, or behind either bronchus by compressing this tube, may cause cough, dyspnoea, and wheezing or croupy respiration. Fibrous or other tumors in the same cavity by their pressure upon the trachea, or œsophagus, may be attended by the same symptoms. The form of intro-thoracic tumor, which is most likely to be mistaken for aneurism, is cancerous deposit in the lung, or anterior mediastinum, particularly if a pulsation is communicated to the tumor. In both cases we may have pain extending to the shoulder, or arm, dyspnoea or stridor in respiration, dysphagia, cough, or



hæmoptysis; in each there is gradual emaciation, the veins of the neck or chest may be enlarged, or œdema of the arm or face may ensue, and a bruit de soufflet may be audible, with dulness on percussion, and bronchial respiration over some part of the chest.

Cancerous deposit in the lung is, however, very rare compared with aneurism, and in its early stage it bears a greater resemblance to phthisis than to aneurism, commencing usually with cough and expectoration, pain in the chest, dyspnoea or hæmoptysis. While in its advanced stage there are usually indications of cancer in some other part or organ, the dulness on percussion is more decided and more extensive than in aneurism, and the situation in which it is most marked is different in the two diseases. Again, the side upon which the cancerous deposit is seated eventually becomes contracted, and bronchial respiration and bruit de soufflet are more generally present in cancer. Emaciation is more pronounced, the skin usually presents the peculiar anœmic appearance of cancer, and the veins upon the surface of the chest are more frequently varicose in cancer of the lung than in aneurism.

When an impulse is communicated to the cancerous mass, or to any tumor lying upon the aorta, it is, as M. Guerin observes, always single, while the aneurismal impulse is always double. Another distinguishing mark to which M. Guerin alludes, is that an aneurism is fixed and immoveable, no matter what position the patient is placed in, owing to the adhesions it contracts, whereas other tumors may be displaced somewhat by altering the posture. This, however, applies rather to abdominal than thoracic tumors.

In cases of empyema it may happen that an impulse is communicated to the sac by the displaced heart, but this never could be mistaken for aneurism. When the matter of empyema makes its way through the costal pleura and forms an external tumor, if an impulse is communicated to it, it may simulate aneurism. This, the pulsating form of empyema, has been well described by Dr. Robert M'Donnell; it is, however, readily distinguished from aneurism by the presence of other signs of empyema, and by the absence of other signs of aneurism, and usually also by the situation at which the tumor presents.

The *complications* of thoracic aneurism may occasionally increase the difficulty of the diagnosis; the disease is however generally met with in subjects who are otherwise healthy, and its



complications are not numerous. Of those which I have met with, viz., fatty deposit on the heart, fatty degeneration of its tissue, hypertrophy of the walls of the left ventricle, and dilatation of its cavity, paralysis and phthisis, the latter is the only one which I have found to increase the difficulty of the diagnosis, the others in no way influenced it. The first to call attention to the complication of thoracic aneurism with phthisis was, I believe, Sir P. Crampton; it must be rather rare on the continent, as Rokitansky says: "spontaneous aneurism never exists in combination with tuberculosis."\*

#### TREATMENT OF THORACIC ANEURISM.

The treatment of thoracic aneurism may be either palliative or curative. The majority of cases admit only of palliative treatment; and we must generally be content with endeavouring to alleviate the most distressing symptoms, or with the employment of such means as we may hope will retard the progress of the disease. Indeed, so utterly hopeless is the disease generally considered to be, and so little benefit is expected from other than palliative treatment, that medical men have occupied themselves much more in observing its progress, and in determining its symptoms, than in following out any line of treatment with the object of endeavouring to effect a cure. Yet we know that nature, in some rare instances, has brought about a spontaneous cure; and, I think, there can be little doubt that if our efforts were directed more frequently to this object we should arrive at results which would give us confidence to persevere.

The indications to be held in view, in the treatment of thoracic aneurism, appear to me to be:

1. To diminish the distending force of the blood from within, by which further enlargement of the sac will be prevented, and it will be placed under a favourable condition to contract.

2. To endeavour to strengthen the parietes of the sac by encouraging the gradual deposition in its interior of the fibrin of the blood which passes through it, by which the risk of its rupture will be diminished.

3. To endeavour to maintain the continued deposition of fibrin in the sac until it is filled and no longer permits the entrance of blood.

\* Vol. iv. Syd. Soc. Trans. p. 295.

4. To bring about these results without deteriorating the quality of the blood, or diminishing too much the patient's strength.

The immediate cause of the distension of the aneurismal sac and of its gradual enlargement, lie, of course, in the impetus communicated to the blood by the systole of the left ventricle: we cannot here, however, as in aneurism seated in an extremity, take off the distending force of the blood by compressing the artery between the sac and the heart; this can only be indirectly accomplished by diminishing the force with which the blood is propelled by the left ventricle, and by lessening the amount of this fluid passing through the sac; which, if we can succeed in accomplishing, the aneurismal sac will be prevented from enlarging, and if the distending force of the blood can be much lessened, the sac will become diminished in size in proportion.

If we look to the manner in which nature brings about the cure of *external* aneurism, we find that this takes place in several distinct ways; but no matter how it is accomplished, the artery at the point from which the aneurism springs is almost invariably obliterated at the same time. In aneurism of the thoracic aorta, on the other hand, there is but one mode in which the disease can be cured—viz., by the gradual deposition of the fibrin of the blood in layers within the sac until it is filled up; and the artery is never obliterated at the site of the aneurism, for an obvious reason.

If, then, we hope to succeed in curing aneurism of the aorta, we must watch the mode in which nature brings it about, and we must endeavour to imitate it.

When a spontaneous cure of aneurism of the aorta takes place, it is always, as I have said, by the gradual deposition of the fibrin of the blood in layers within the sac until it is filled up, and no longer permits of the entrance of blood. In order, therefore, to favour such a result, we must endeavour to diminish the force and the frequency with which the blood traverses the aneurismal sac, and we must likewise endeavour to lessen the quantity transmitted through it. In external aneurism these objects can be effected by compressing the artery upon the cardiac side of the aneurism; in aneurism of the aorta we can only effect them indirectly by acting upon the general circulation.

As exercise has the effect of quickening the heart's action, and

rest has an opposite effect, and tends to quiet the circulation, absolute rest of body and mind are indispensable as preliminaries. Again, we know that the heart's action is less rapid in the recumbent than in the erect or sitting posture; it is essential, therefore, that the patient should preserve the recumbent posture, and be confined to bed during a portion at least of the period that the treatment lasts.

In order to diminish the amount of blood, or to reduce the force of the circulation, bloodletting has been more frequently employed than any other remedy, and probably is the oldest which has been used with this object. Now, copious or frequent bloodletting has serious disadvantages; it impoverishes the blood, removing the fibrin, the very substance, the presence of which in this fluid is essential to the cure. While if bloodletting is combined with very low diet, according to the plan long since recommended by Albertini and Valsalva, there is no possibility of the fibrin being renewed. Again, frequent and copious bloodletting not only impoverishes the blood by removing fibrin, but has the further ill effect of increasing the frequency of the heart's action and hurrying the circulation, thus occasioning the very contrary results to what we desire.

The plan of treatment, termed that of Valsalva, is the one preferred by Laennec, by Bertin, and by Bouillaud. Dr. Hope, while he objects to the copious and frequent abstraction of blood advocated by the foregoing authors, recommends it within certain limits. "The patient should in the first instance (he observes) be pretty copiously bled, from twelve to twenty ounces being drawn according to the age and strength. After this it will generally be sufficient to abstract six or eight ounces every three to six or more weeks; the quantity being the larger and the interval shorter in those who are robust and plethoric, and who speedily reproduce blood."

It appears to me that there are very few cases in which general bleeding is required; while copious or frequent venesection is calculated to do harm rather than good, and to retard or prevent rather than promote a cure:

1. By removing the *fibrin*, an essential agent in the cure.
2. By producing a watery state of the blood, when blood, rich in the solid ingredients, is required.

3. By tending to quicken the circulation, when it is desirable that it should be rendered slower; and

4. By unnecessarily debilitating the patient, and inducing an anæmic condition of the system.

In order to diminish the watery portion of the blood, purgatives, particularly those which cause fluid evacuations, are recommended by some—by others diuretics are given with the same object. “Occasional purgation, continued for a week or ten days at a time, may (Dr. Hope observes) be resorted to with great advantage after bloodletting, as it keeps down the quantity of the blood, without depriving it to the same degree of its fibrin. In this view the purgatives which produce aqueous evacuations are the most suitable. The neutral salts will suffice for ordinary occasions; but, when a powerful effect is required, nothing is comparable to elaterium, by which two or three pints of serum or more may sometimes be drained away in twenty-four hours. Jalap and bitartrate of potash have in a less degree the same effect.”

The advantage expected from the frequent use of saline or hydragogue cathartics, is to render the blood more dense, by removing, to a certain extent, the watery portions. It is, undoubtedly, of importance for the success of the treatment that the blood should be as rich as possible; but would it not be more consistent with common sense, instead of endeavouring to remove the more watery parts of the blood by copious and active purgation, to restrict the patient to so small a quantity of drink, that hydragogue cathartics or diuretics would be unnecessary? As every particle of fluid swallowed must pass through the current of the circulation before it can be removed by purgatives from the alimentary canal, or before it can be excreted by the kidneys, it would seem to be a much more direct and rational proceeding to diminish the drink to the smallest amount, rather than to allow the patient to drink, and then to endeavour to remove it by cathartics or diuretics. Besides, the action of purgatives, frequently repeated, is calculated to distress and weaken the patient; while their operation must in some degree quicken the circulation, and prevent the perfect repose so essential towards a cure.

In order to lessen the frequency of the heart's action, or to lower the circulation, digitalis has been frequently employed. “Digitalis is said to be useful in the treatment of aneurism, by

enfeebling and retarding the action of the heart and arteries, and thus promoting the stagnation of the blood within the sac." As this medicine, however, in order to produce any effect, must be persevered in for some considerable time, it is likely to prove a dangerous remedy, owing to its cumulative property; indeed, in the form of disease which we are considering, it is even more dangerous than in ordinary cases, and in my mind ought never to be employed.

In order to favour what is called "coagulation in the aneurismal sac," different medicinal substances have been recommended. Dr. Hope advocates the use of the acetate of lead, in small doses frequently repeated; "it may be given (he observes) occasionally when digitalis disagrees, or where the patient tires of that remedy, or takes a prejudice against it." Others recommend the preparations of iron with the view of improving the state of the blood. There can be no objection to the employment of the latter medicine, but the administration of acetate of lead is calculated to prove rather detrimental than otherwise; and as aneurism of the aorta is not cured by "coagulation in the aneurismal sac," its administration with this object, even if it had the power of causing coagulation, is absurd. Indeed, if the blood were to become coagulated in the sac of a thoracic aneurism, it would place the patient's life in imminent peril; as the clot or coagulum would sooner or later be driven by the current of the blood out of the sac into the aorta beyond it, which, according to its size and firmness, it would fill or obstruct, and such an impediment to the circulation would be occasioned, as quickly to cause the patient's death.

The plan of treatment which appears to me to be best calculated to fulfil the indications we have in view, is almost essentially dietetic. It consists in limiting the patient, for a given period, to the smallest quantity of fluid possible—in diminishing likewise the solid aliment—in confining the patient at the same time to bed—and endeavouring to maintain the mind in as tranquil a state as possible. I am neither an advocate for bleeding or purgatives, for diuretics or digitalis, or any of the other medicines which have been used in this disease, with the exception of opium, and this only when sleep is prevented by pain.

By confining the patient to the horizontal posture, the circulation is tranquillized, and the heart's action becomes slower. When

this is combined with a small quantity of solid nutriment, and a still smaller quantity of liquid, the heart's action will become slower, and the pulse compressible, small, and soft. Its effect upon the blood will be to render this fluid thicker, as the watery portions are excreted by the kidneys and skin. Thus less blood will pass through the aneurismal sac, it will be transmitted with less force, and in a diminished stream, while its quality will be improved; all which circumstances are favourable to the deposition of fibrin in the aneurismal sac; and as the muscles are not exercised, there is no waste of fibrin in supplying them.

The diet constituted, we know, an important item in the treatment of aneurism advocated by Albertini and Valsalva, where a very low diet was combined with copious and frequent bloodletting. Some modern writers, likewise, recommend certain restrictions in this particular, but they have been content with general directions under this head. I do not think we shall be able to effect much unless precise directions as to the exact quantity and kind of food are laid down. I would limit the patient to three meals a day, the morning and evening meal to consist of two ounces of liquid, and four of solid nutriment; the mid-day meal of four ounces of liquid, with from four to six ounces of solid. The liquid may consist of milk or tea, the solid of bread, and the mid-day meal, of bread and meat in equal quantity. No deviation from this dietary should be permitted, and it must be persevered in for a fixed period, six weeks or a month at least, when it may gradually be improved. If the patient is weighed on commencing it, and this is done occasionally afterwards, we shall have a guide as to the advisability of continuing it, or of improving it.

This plan of treating aortal aneurism is not proposed on purely theoretical grounds. I have employed it in several cases with beneficial results. It is applicable not only to aneurism of the thoracic and abdominal aorta, but to all those cases where the sac springs from a branch of this vessel, and is beyond the reach of surgical interference, as aneurism of the innominate, of the subclavian, and of the carotid at the root of the neck, as well as aneurism of the common iliac. I may add that I have employed it, but for a shorter period, with much advantage as a preliminary to the application of compression in popliteal aneurism.

In a mode of treatment such as this, success will depend in a

great measure upon the perseverance with which it is carried out. It is therefore necessary that the patient should be made aware of the dangerous nature of the disease under which he labours; as in such a case he will be more likely to submit cheerfully to the restrictions imposed upon him. Indeed, unless he co-operates with us in carrying out the treatment fully and fairly, it can never be effectually maintained.

It might be objected that the tendency of so very restricted a diet is to produce a state of anæmia; but it appears to me that anæmia is not much to be dreaded, if bloodletting is not employed. When death has been the result of inanition, owing to stricture of the œsophagus, or to the pressure from without of an aneurismal or other tumor, we do not find that anæmia precedes it.

The situation of the aneurism, or the point from which it springs, its size, and the nature and importance of the parts which are compressed, will exercise an influence upon the results of treatment. If the aneurism had attained a very large size when first seen; if it springs from the upper part of the arch, and forms a prominent tumor in the neck, above the sternum, or above the right or left clavicle, the progress of the disease is often rapid, and curative treatment has less influence. As long as an aneurismal sac is confined within the bony walls of the thorax, the parietes of the sac receive support from the parts in the vicinity with which the sac forms adhesions, as well as from the walls of the thorax, by which its very rapid dilatation is prevented; but when it rises out of the cavity of the thorax, no longer having this support, the sac yields to the distension from within, and the tumor usually increases rapidly.

Again, although the aneurismal sac be confined within the bony parietes of the chest, if the sac has formed adhesions with the trachea, or with the œsophagus, compresses these parts, and materially interferes with their functions; or if the par vagum, the recurrent, or the phrenic nerves are involved in the tumor and compressed or stretched by it; or if the sac has formed adhesions with the bodies of the vertebræ, and has caused erosion of the bone, or with the descending cava, so as to compress it and interfere with the return of the blood to the right auricle, the same advantage cannot be expected from curative treatment as where



none of these parts are involved. Still if, by means of treatment, the distension can be lessened, or taken off from the sac, the latter will probably diminish in size, the injurious pressure upon the parts in the vicinity will be diminished, and unless permanent injury has been occasioned by the pressure, the parts may be restored to their former condition.

## CONCLUSIONS.

The following conclusions under this head may, I think, be deduced from what precedes :

1. That thoracic aneurism is not necessarily an incurable disease.
2. That it appears to be more amenable to curative treatment than is ordinarily supposed.
3. That treatment ought, as a general rule, to be specially directed to this object.
4. That when a spontaneous cure occurs, it is always by the gradual deposition of the fibrin of the blood in layers within the aneurismal sac until it is filled up.
5. That if we hope to succeed in effecting a cure, it must be by imitating the mode in which nature brings this about.
6. That in order to favour the gradual deposition of fibrin, we should aim at diminishing the mass of blood, and lessening the strength and rapidity of the current through the aneurismal sac.
7. That this can only be indirectly accomplished by acting upon the general circulation.
8. That neither bleeding, purgatives, diuretics, digitalis, nor the various other remedies which have been employed in this disease, can be depended upon for producing these effects.
9. That an extremely restricted diet, particularly in fluids, continued for a certain time, appears to have the effect of rendering the pulse small, compressible, and slow, and at the same time of diminishing the mass of blood.
10. That this method of treatment, to prove effectual, must be steadily and perseveringly carried out, and must be continued until a decided impression is made upon the disease.



11. That it is adapted not only to aneurism of the thoracic and abdominal aorta, but to aneurism in any of the immediate branches of these vessels.

12. That if employed as a preliminary to compression, in popliteal or femoral aneurism, pain will be diminished, and the duration of the treatment considerably abridged.

**END.**













